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THIRD SERIES FOUNDED BY EDWARD JACKSON

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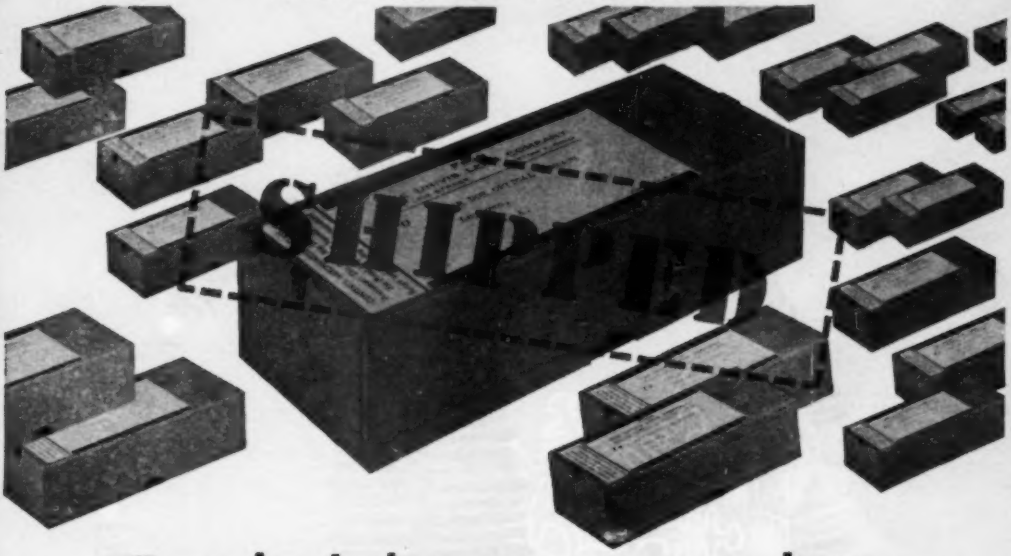

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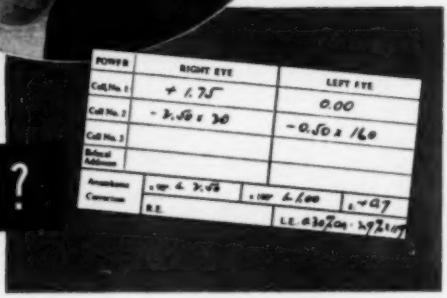

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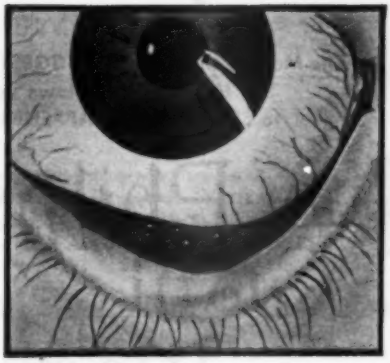

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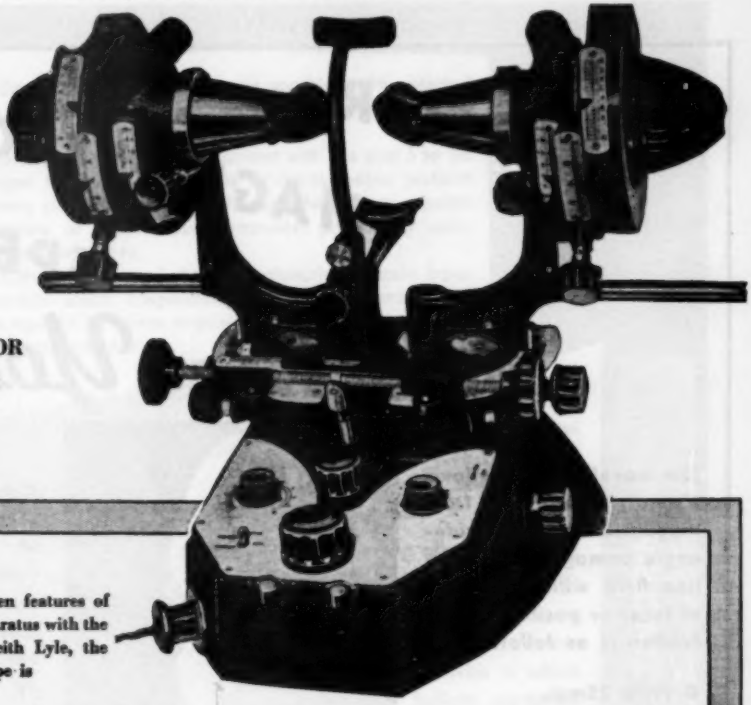

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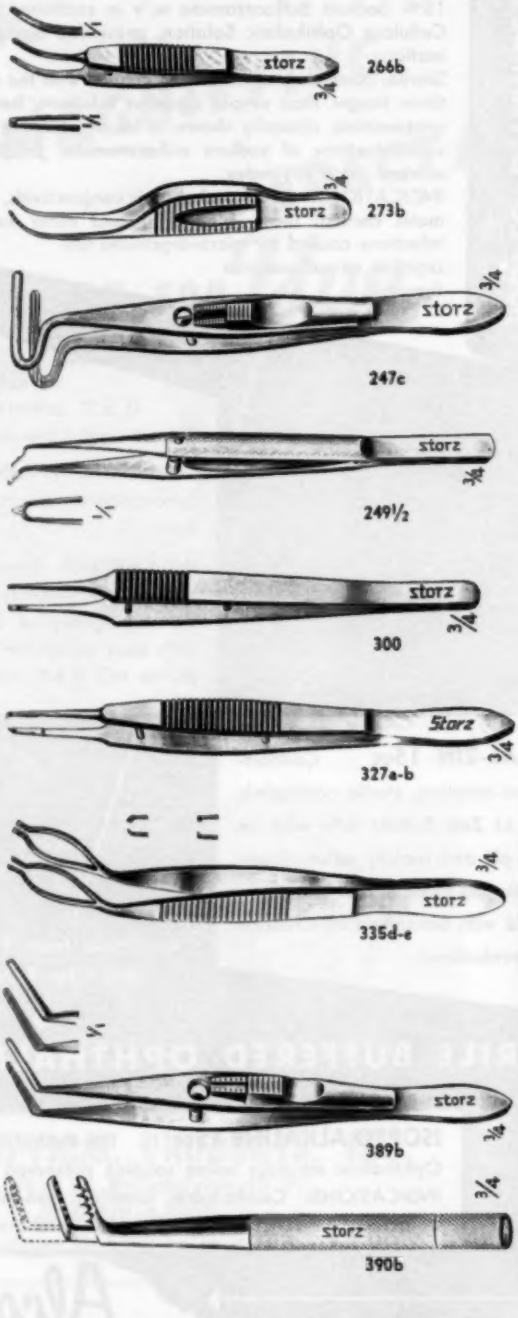

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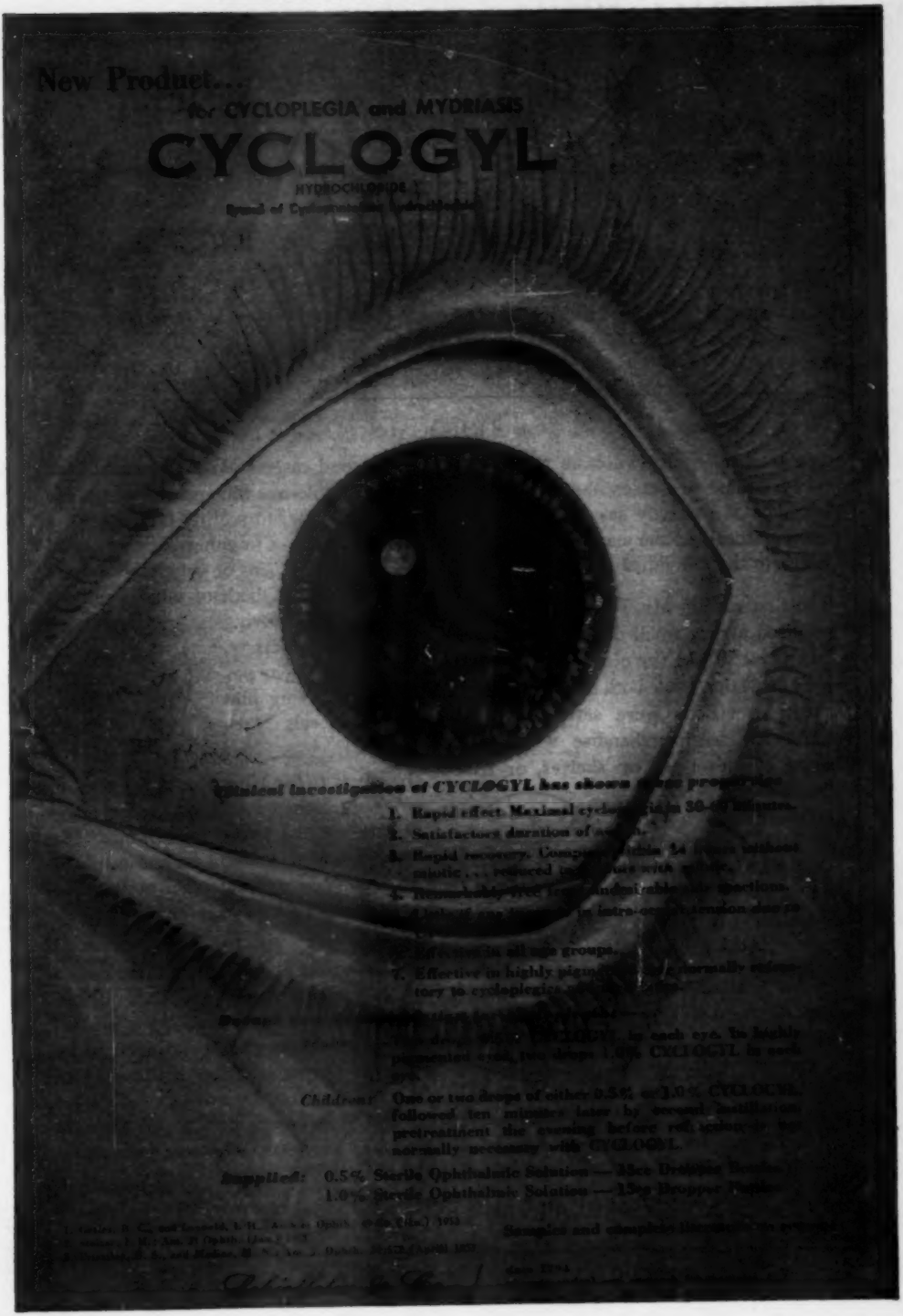
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We are presenting two cases this month because these two cases illustrate the importance of correctly selecting, from the many trifocals on the market, the one best suited to the individual case.

The first case is Mr. D, a druggist, who required a very small distance correction and a +2.50 add for near. Mr. D's complaint was that while both the distance and the near visions were satisfactory, he could not see merchandise which was above eye level and on shelves, or read the labels on prescription bottles which were similarly located. Mr. D's problem was solved by giving him "baseball" trifocals (see figure 1).

Fig. 1

In the "baseball" trifocal, the bottom segment is for reading, the central portion for distance and the upper segment is the intermediate. In the case of Mr. D the intermediate add was +1.25. The "baseball" trifocal is used for storekeepers, mail clerks, librarians and all others who need an above eye level intermediate segment.

The second case is Mr. W, an architect, whose trouble was blue print reading. He complained that to see the far edges of a blue print he had to either back away and use the distance lens or get closer and use the reading lens, both of which methods were most annoying and unsatisfactory. Mr. W's problem was solved by giving him ultex trifocals (see figure 2).

Fig. 2

The ultex trifocal has the lower portion for reading, the central portion for intermediate and the upper for distance. The ultex trifocal is excellent for use at a desk, for draftsmen, and for other occupations that require below eye level intermediate vision.

The two cases above illustrate the use of but two of the many trifocals available. It is well to know about all of them because the success of the fitting depends upon the proper selection of the segment size and position. Drawings of different trifocals together with data on segment sizes and shapes will be sent you on request.

"IF IT'S A LENS PROBLEM, LET'S LOOK AT IT TOGETHER"

AMERICAN JOURNAL OF OPHTHALMOLOGY

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2. McLaurin, J. W.: Laryngoscope, 61:66, Jan., 1951.
3. Craig, P. E.: Jour. Oklahoma Med. Assn., 42:523, Dec., 1949
4. Lott, James E.: Air University, USAF School of Aviation Medicine. Report No. 1, Project No. 21-32-036, p. 4.

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AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 36 DECEMBER, 1953 NUMBER 12

OCULAR MANIFESTATIONS OF DISTURBANCES IN CALCIUM METABOLISM*

THE NINTH SANFORD R. GIFFORD LECTURE

FRANK B. WALSH, M.D., AND ROBERT G. MURRAY, M.D.
Baltimore, Maryland

The honor accorded me in the invitation to deliver the Gifford Lecture is deeply appreciated. However, since my acceptance I have become increasingly aware of the excellence of the presentations of other years and the tradition that this lecture calls for new information. Such disconcerting reflections and an awareness that I have nothing new to offer are responsible for what is an unusual approach to a clinical study.

For years I have been intrigued by the diversity of cases in which calcium is an important consideration and have been confused by laboratory reports and clinical findings which, in some instances, have seemed contradictory. With the hope of remedying the situation, at least in part, for my own edification, I have re-examined much of the clinical material which is available to me.

From this study an attempt has been made to correlate certain basic facts regarding calcium in human metabolism with selected groups of clinical cases which are of interest to ophthalmologists.

I. BASIC CONSIDERATIONS

Regarding the importance of calcium in body processes Best and Taylor stated: "Calcium is an indispensable material: it is a constituent of all animal fluids and solid tissues and plays an important role in a number of physiologic processes and conditions. The most obvious of these are (a) coagulation of the blood, (b) formation of bone, (c) cardiac rhythmicity, (d) maintenance of neuromuscular excitability, (f) membrane permeability."

BLOOD CALCIUM

The blood plasma contains almost all the calcium in the blood. When blood clots, the calcium is principally present in the serum, only a negligible quantity remaining in the clot. There are two forms of calcium in the blood: (1) Nondiffusible, which is bound to the protein in the serum (45 percent), and (2) diffusible, present as carbonate and phosphate and ionized in by far the greater part (55 percent).

A normal report on blood serum calcium is 9.0 to 11.5 mg. percent (per 100 cc.); whereas, something below 9.0 mg. percent indicates hypocalcemia and something over 11.5 mg. percent indicates hypercalcemia.

It is changes in ionized calcium which are responsible for alterations in muscle and nerve excitability. Furthermore, such ionized (diffusible, ultrafilterable) calcium to be utilized reaches and leaves the capillaries where its concentration is between 5.0 and 7.0 mg. per 100 cc. (Howard).

There is a remarkable constancy as regards the amount of calcium in the blood serum of normal individuals. Such homeostasis merits consideration before mention is made of relationships which may exist between serum calcium and inorganic phosphorus in normal and abnormal states.

Howard has suggested recently that the bony skeleton is physiologically responsible

* From the Wilmer Institute of The Johns Hopkins University and Hospital. Presented at the clinical conference sponsored by the Chicago Ophthalmological Society, Chicago, February 14, 1953.

for stability of serum calcium. In supporting this proposition he has drawn attention to several observations:

1. The bones and teeth are the only possible sources for considerable amounts of calcium.

2. Normocalcemia or rapid return to it is seen in experimental animals subjected to deficiency or excess of calcium in diet.

3. The intestinal tract and the kidneys seem to play minor roles as regards calcium balance.

4. Recent studies by Hopkins and others show that normal blood serum is not saturated with calcium. The addition of calcium (up to a saturation of 20 mg. percent) produces a uniform increase in the diffusible (ionized) and nondiffusible (protein-bound) calcium.

As regards phosphorus in the blood serum the mechanisms are more complicated. Probably the skeletal supply of phosphorus is of principal significance but there is ample evidence, not gone into here, that the kidney plays an important role as regards the level of serum phosphorus. Inorganic phosphorus of blood serum varies from 4.5 to 6.0 mg. per 100 cc. in normal children and from 3.3 to 4.0 mg. per 100 cc. in adults.

The spinal fluid contains ionized calcium 4.5 to 6.0 mg. per 100 cc. This is almost invariably constant and not altered by hyper- or hypocalcemia.

It is generally well known that an increased serum calcium level is often associated with a decreased serum phosphorus and decreased serum calcium with increased serum phosphorus. Such findings have suggested that, within the serum itself, serum calcium and phosphorus have a reciprocal influence. Howard's proposal is to the effect that changes in calcium and phosphorus levels in the serum reflect cellular changes and a physiologic response to such changes.

Calcium balance

This is dependent upon ingestion, absorption, and excretion of calcium. It is absorbed largely in the small intestine and is excreted principally in the feces and in lesser amounts in the urine.

It is an important constituent of bone where it is not inert but may be withdrawn particularly under the stimulus of there being insufficient calcium for the requirements of other tissues. In addition to such physiologic mobilization of calcium from bone, demineralization may result from abnormal activity of the parathyroid glands, the administration of parathyroid extract, and from toxic dosages of vitamin D.

Calcium retention (positive balance) associated with deposition in bones occurs during growth, with acromegaly, and after a period of calcium starvation. Loss of calcium (negative balance) is seen in rickets, sprue, osteomalacia, and hyperparathyroidism during calcium deficiency and lactation (Best and Taylor). Vitamin D provides a positive balance in some cases of rickets and osteomalacia.

Alkaline phosphatase

Phosphatase is an enzyme which is active in the growth of bone. Alkaline phosphatase of the blood serum is an estimation which has value in diseases characterized by changes in the bones. The phosphatase activity of the serum (Bodansky method) is 5.0 to 15 units per 100 cc. during childhood; after puberty it varies between 1.5 and 4.0 units per 100 cc. Usually phosphatase is increased as a result of active bone disease, as for example in Paget's disease, osteitis fibrosa cystica, myeloma, leukemia, rickets.

The parathyroids: Hypoparathyroidism: Hyperparathyroidism

The *parathyroid* glands are responsible in large part for the maintenance of normal calcium and phosphorus metabolism. Hypoparathyroidism produces hypocalcemia and hyperparathyroidism is characterized by hypercalcemia.

Hypoparathyroidism occurs rarely as a temporary dysfunction in the newborn; it is

characterized by tetany. Idiopathic hypoparathyroidism is seen in association with Addison's disease in some instances. Such an association may be fortuitous. Idiopathic hypoparathyroidism usually occurs in children. As the eponym suggests its cause is not known. Hypoparathyroidism in adults usually arises as a result of the accidental removal of the parathyroid glands during thyroidectomy. The resultant tetany is termed "parathyroprivic."

Pseudohypoparathyroidism is a rare disorder. It has the same clinical and laboratory findings as occur in hypoparathyroidism but with pseudohypoparathyroidism there is absence of response to parathyroid extract. Otherwise stated, in pseudohypoparathyroidism there is an absence of end-organ response. The parathyroid glands secrete normal amounts of hormone; it has been suggested that the renal tubules which control the reabsorption of phosphorus do not respond to parathormone.

Ellsworth and Howard devised a test which makes it possible to differentiate between hypoparathyrodism and pseudohypoparathyroidism. They showed that in the normal individual and in the hypoparathyroid patient a test dose of parathyroid hormone produced phosphorus diuresis followed by a fall in the serum phosphorus and later a rise in serum calcium.

Parathormone fails to produce phosphorus diuresis in pseudohypoparathyroidism. However, A. T. 10 (Hytakerol) and vitamin D are effective in this metabolic defect.

Hyperparathyroidism is of two types, primary and secondary.

Primary hyperparathyroidism is associated with edenoma of the parathyroids or with parathyroid hyperplasia. It is seen almost exclusively in adults.

Secondary hyperparathyroidism is, for the most part, associated with renal disease. The present concept regarding its origin seems to explain certain of the signs of so-called "renal rickets."

According to Wilkins, "renal rickets" is a misnomer and the affection should be termed chronic renal insufficiency with phosphate retention. In children with congenital abnormalities (or defects) of the kidney or with any form of chronic renal disease there may be elevation of nonprotein nitrogen and phosphate retention as a consequence of the renal damage.

What then is secondary hyperparathyroidism?

Seemingly, as the result of the intrinsic relationship between serum calcium and phosphorus (high calcium: low phosphorus-low calcium: high phosphorus) the low serum calcium (induced by the hyperphosphatemia) excites a hyperplasia of the parathyroids. This is secondary hyperparathyroidism.

Regulation of the serum calcium and phosphorus

Although the parathyroid glands have a primary function in controlling the calcium and phosphorus other influences may alter the amounts of these substances in the blood. Wilkins stated in part:

"It is apparent that the regulation of the concentration of serum calcium and phosphorus depends upon the absorption of these elements from the intestine, their excretion through the kidneys or bowel, and their mobilization from or deposition in the bones. . . . Vitamin D deficiency or steatorrhea may interfere with absorption from the intestine, or renal disease may prevent normal excretion. Bone disease, atrophy of disuse, or acidosis may cause excessive mobilization of calcium and phosphorus. The body attempts to compensate for such abnormalities so as to maintain a normal concentration in the serum. If there is a deficient absorption of calcium less is excreted, or there may be mobilization from the bones. If there is excessive demineralization of bone, larger amounts of calcium and phosphorus are excreted in the urine and renal calculi or hephrocalcinosis may result. However, the capacity of these mechanisms to compensate

may be exceeded so that subnormal or excessive concentrations of the blood calcium or phosphorus may result."

Vitamin D and related substances—calciferol (viosterol) and dihydrotachysterol (A. T. 10) among others—have a profound effect on calcium and phosphorus metabolism but the mechanisms through which they function are not known with any certainty. It is established that vitamin D and viosterol are helpful in rickets, whereas A. T. 10 which has little or no effect on rickets has a pronounced capacity to elevate the serum calcium level. Whether vitamin D increases absorption of calcium from the bowel or promotes deposition of calcium and phosphorus in the bones is undetermined (Best and Taylor).

II. Conditions associated with hypercalcemia

Hypercalcemia is associated with many clinical affections. Most frequently it arises from vitamin-D intoxication or from hyperparathyroidism. Also it may characterize sarcoid, multiple myeloma (20 percent of cases —Baird and Keck), osteolytic metastatic carcinoma particularly when sex hormone is being used (Hermann et al.; Prestin et al.), immobilization for fractures or even prolonged bed rest (usually there is an associated osteoporosis), increased intake of milk and alkalis in ulcer therapy (Burnett et al.), and in beryllium intoxication (Cogan).

According to our experience the ocular evidences of hypercalcemia have been observed usually in hyperparathyroidism associated with adenoma of the gland and vitamin-D intoxication, infrequently in sarcoid.

Hyperparathyroidism

Primary hyperparathyroidism has an interesting symptomatology from the standpoint of what is known as regards calcium metabolism. There is elevation of the serum calcium, usually to above 12 mg. per 100 cc.; inorganic phosphorus is diminished to below 2.5 mg. per 100 cc.; there is elevation of the alkaline phosphatase to above 7.0 to 8.0 units per 100 cc. (in less than 50 percent of cases seen in the Johns Hopkins Hospital—J. E. Howard).

Pain in the back and extremities is an infrequent early symptom and in approximately 20 percent of our cases there is decalcification and cystic changes in the bones (osteitis fibrosa cystica). Such changes are due to withdrawal of calcium from the bones.

The urine contains excess calcium. There is wasting of muscles and loss of tendon reflexes as well as loss of electrical excitability of the muscles; such changes are probably directly due to the hypercalcemia. Deposition of calcium occurs rarely in soft tissues (so-called metastatic calcification) and this accounts for the ocular symptomatology which is seen in a minority of cases.

The hypercalcemia accounts for kidney stones. The kidney involvement may produce uremia and this may be responsible for death.

Vitamin-D intoxication

This intoxication occurs not infrequently. Ocular signs have been present in a majority of our cases. I have examined the patients described by Howard and Meyer in their paper written in 1948.

In all 10 cases included by Howard and Meyer in their Table 1, hypervitaminosis D had arisen as the result of large doses of various preparations used in the treatment of arthritis (Ertron; Deltalin; Davitin; Darthronal). The largest daily dose was 600,000 I.U.; the smallest daily dose was 150,000 I.U. Duration of therapy before onset of symptoms was extremely variable with a minimum of two and a maximum of 18 months.

The symptomatology was described by Howard and Meyer as follows:

Anorexia, nausea, and vomiting were early symptoms in most cases. Invariably

there was complaint of weakness, fatigue, and lassitude. Weight loss was prominent. Polyuria, polydipsia, or nocturia were present in most (70 percent) of their patients.

In all cases there was evidence of renal damage at the time the diagnosis was made; total protein and AG ratio were normal (ratio tested in seven). The urine invariably contained calcium (Sulkowitch test).

Inorganic phosphorus in the blood serum varied between 3.0 and 5.2 mg. per 100 cc. (usually above the range of normal but not markedly so in the face of the nitrogen retention). The urine contained small amounts of albumin in seven of 10 cases.

The optic fundi were essentially normal.

After withdrawal of the vitamin D preparation and "forced fluids," as well as a diet low in calcium, a subjective improvement was obtained in all cases within two to eight weeks. Serum calcium in all but two instances diminished within a month. In some cases follow-up reports showed decrease of the serum inorganic phosphorus. In some instances there was improvement in kidney function.

Metastatic calcification of the cornea or conjunctiva was present in eight of 10 cases. Lesions of white-yellow color were present on the lips (fig. 1) and under the nails (fig. 2) of their Case 4; biopsy of the skin in this individual showed senile elastosis with deposits of calcium in the midcorium. Such calcification is reversible at least in part in the eyes and elsewhere.

Fig. 1. (Walsh and Murray). Deposits of calcium on the lips in hypervitaminosis D. Such deposits disappear ultimately after the vitamin is discontinued (from Howard and Meyer).

Fig. 2 (Walsh and Murray). Deposits of calcium under the nails (Howard and Meyer—Case 4).

Sarcoidosis

Sarcoidosis is, in some instances, associated with hypercalcemia. With sarcoidosis there may be hyperproteinemia and inversion of the albumin: globulin ratio (normal readings: total protein 6.0 to 8.0 gm. percent; albumin 4.0 to 5.0 gm. percent; globulin 1.0 to 3.0 gm. percent).

Harrell and Fisher found hypercalcemia in six of their cases of proven sarcoidosis. The serum phosphorus levels were normal in all. They did not think the hypercalcemia could be explained on the basis of the hyperproteinemia but felt it probably reflected changes in the bone marrow.

It is to be noted that serum phosphatase is elevated in all active cases and that bone-marrow activity may exist when bony changes cannot be detected in X-ray films.

Ocular signs of hypercalcemia

It has been generally well known for years that long-continued intraocular inflammation may precede the development of band-shaped keratitis and that in some such cases flakes or sheets of calcium may be peeled from the cornea. It has not been well known that calcification of the cornea and conjunctiva may develop on the basis of hypercalcemia in the absence of any ocular inflammation. What is more important is that such "metastatic" calcification may be an early sign of hypercalcemia and is seen frequently as an isolated sign of the disorder.

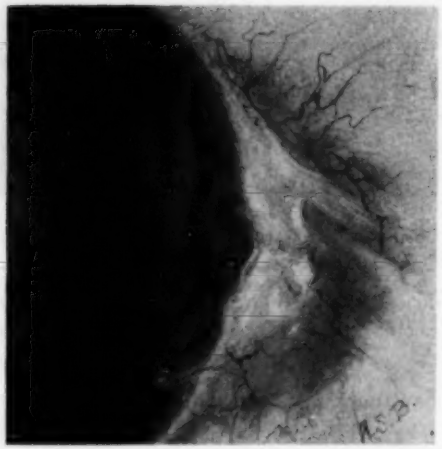

Fig. 3 (Walsh and Murray). Slitlamp appearance of hypercalcemia affecting the cornea (Walsh and Howard). The corneal appearance shown here is more specific than changes in the conjunctiva which consist of glinting particles.

Calcification of the conjunctiva and cornea was described in this country in 1947 as an evidence of hypercalcemia (Walsh and Howard). Also in that year Frost, Sunderman, and Leopold described metastatic calcification of the sclera as a result of vitamin-D intoxication. Calcification of the cornea and conjunctiva was further described in 1948 by Cogan, Albright, and Bartter. In 1952, Fleischner and Shaler showed that X-ray visualization of such ocular calcification may be obtained readily.

After having had considerable experience in identifying hypercalcemia through slitlamp examination, I wish to emphasize that the corneal picture is more specific than the conjunctival appearance although either or both may be sufficient to suggest the diagnosis.

The reason for this is obvious. In the conjunctiva there are frequently found, at the corneal edges particularly, accumulations of elastic tissue (pinquecula), pigment deposits, and fibrous tissue. Such abnormalities as these explain the presence of local calcifications not present on the basis of hypercalcemia but as a purely local process.

It may be noteworthy that in our cases of hypercalcemia there has been absence of retinopathy in all but one instance. In that case (W. V. S. 487759), the basic process producing the hypercalcemia was not determined and sarcoidosis was suggested but not proven as the diagnosis; there was moderate hypertension and a choroidal sclerosis of unusual severity.

I have seen several cases in which calcification of the conjunctiva and cornea arose as the result of injudicious treatment of hypoparathyroidism. Calcification of the cornea is rarely seen in very old people in the absence of hypercalcemia.

Grant has discovered an effective treatment for calcific corneal opacities. If epithelium overlies the opacity, the epithelium must be removed. The denuded area is then irrigated for 15 minutes with a sterile 0.01 M solution of EDTA (neutral sodium salt of ethylenediamine tetracetic acid).

We have considered an incomplete band as characteristic of corneal calcification (fig. 3).

Through the courtesy of Dr. David G. Cogan I am able to report a remarkable case in which almost the entire surface of both corneas was calcified.

Case Report. A 38-year-old man, an employee of an insulated wire company, developed frequency of urination, weight loss, and scalp infection eight months before his examination by Dr. Cogan. It was found that he suffered from diabetes mellitus. The diabetes was controlled by 30 units of insulin daily.

Two months after the development of diabetes there was complaint of halos about light, progressive blurring of vision, and glare.

Examination revealed diffuse superficial opacification of both corneas, except for a one- to two-mm. clear zone at the upper and lower limbus regions. The opacities were situated just beneath the epithelium. Despite the opacification, the vision was 20/20 and 20/50, right and left eye respectively.

The conjunctiva contained translucent crystals. The posterior surface of the corneas was clear. The irises were normal. The fundi could be well seen and were normal.

Blood serum calcium was 14.6 mg. percent; phosphorus, 5.5 mg. percent; phosphatase, 17.3 units per 100 cc.; serum cholesterol, 137 mg. percent.

The patient was admitted under the care of Dr. Fuller Albright. The blood chemistries were con-

TABLE 1

OCULAR HYPERCALCEMIA WITH ADENOMA OF THE PARATHYROIDS

Name and Number	Conjunctival Calcification	Corneal Calcification	Remarks
M. D. 386092	Negative	Negative	
L. W. 440159	Negative	Negative	
A. K. 224656	Positive	Positive	
M. L. 366891	Negative	Positive	
H. C. 623930	Negative	Negative	
A. D. 624279	Positive	Negative	
J. R. 400323	Negative	Negative	
R. G. 543335	Negative	Negative	
D. S. 557218	Negative	Negative	
B. B. 575980	Negative	Positive	
G. M. 447323	Negative	Negative	
H. B. 175185	Negative	Negative	
P. L. P. 621251	Negative	Negative	
D. T. 621251	Negative	Negative	
B. M. 104641	Negative	Negative	
M. 204618	Positive	Positive	
F. Z. K. 190342			Calcification sclera at autopsy

SUMMARY: Four of 16 cases showed calcification cornea or conjunctiva or of both.
Other cases of proven parathyroid adenoma not examined with slitlamp—12 cases

OCULAR HYPERCALCEMIA WITH VITAMIN-D INTOXICATION

Name and Number	Conjunctival Calcification	Corneal Calcification	Remarks
J. S. 419594	Positive	Positive	
W. C. 421206	Positive	Positive	
U. G. 466336	Positive	Positive	
D. F. 483743	Negative	Positive	
F. H. 168084	Positive	Negative	
A. W. 195939	Positive	Positive	
J. J. 386002	Negative	Negative	
B. F. 336649	Positive	Positive	Overtreatment for hypoparathyroidism cataracts
E. L. 366421	Positive	Negative	
L. S. 414730	Positive	Negative	
C. B. T. 602225	Positive	Positive	
M. D. 446602	Positive	Positive	
J. B.	Positive	Positive	Courtesy Dr. Iliff
M. M.	Positive	Positive	Courtesy Dr. Trescher
D. M.	Positive	Positive	

SUMMARY: 13 to 15 cases showed calcification of the conjunctiva, or of the cornea or of both. Calcification of the cornea is a more specific finding.

OTHER CASES OF OCULAR CALCIFICATION

Sarcoid	2 Cases	R. N. 481401 R. H. H. 610287
Immobilization and hypercalcemia	1 Case	K. Z. (BCH) 167178
Alkali treatment peptic ulcer	1 Case	M. H. 632931. It is possible that this patient has a parathyroid tumor. He is presently taken off alkali treatment for peptic ulcer.

firmed. Symptoms of kidney stones were denied. There was no history of consumption of vitamin D.

X-ray films of the bones showed no evidence of demineralization. Flat plates of the pelvis showed kidney stones; lungs normal; bone-free X-ray films of the eyes failed to show calcium but, with von Kossa stain, it was shown that there was calcium present beneath the epithelium of one cornea so tested. The patient had a large liver and spleen. A liver biopsy showed giant cells, epithelioid cells, and lymphocytes which strongly suggested sarcoidosis.

It seems probable that the diabetes is caused by sarcoid infiltration of the pancreas.

The patient is now in hospital for cortisone therapy.

My experience with ocular evidences of hypercalcemia is shown in Table 1.

III. Conditions associated with hypocalcemia

Hypocalcemia is a significant finding in hypoparathyroidism, steatorrhea, rickets, and may occur in nephrosis and hypoproteinemia. It develops after prolonged ACTH therapy (Levin et al.). In hypoparathyroidism and secondary hyperparathyroidism the low serum calcium is combined with high phosphorus; whereas, in other conditions, the low calcium is associated with normal or decreased serum inorganic phosphorus (Wilkins).

Tetany

Tetany develops on the basis of reduction of the ionized calcium. Thus it is possible for the total serum calcium to be within normal levels and tetany to develop on the basis of reduction of the ionized (diffusible) fraction. There may exist a reduction in total serum calcium as in nephrosis but absence of tetany because the ionized calcium is present in normal amounts.

Hypocalcemia associated with tetany is characterized by increased excitability of muscles and increased tendon reflexes which is precisely the opposite to what occurs in hypercalcemia.

The remainder of this section concerns hypoparathyroidism which may be divided into three principal types: (1) Idiopathic or primary; (2) pseudohypoparathyroidism; (3) acquired or postoperative hypoparathyroidism. Since pseudohypoparathyroidism is of infrequent occurrence it receives little further attention.

Idiopathic hypoparathyroidism

As the name implies this entity is of unknown origin; seemingly it is dependent upon intrinsic factors in the parathyroid glands.

According to Steinberg and Waldron only 52 cases, including one case they observed, have been reported up to 1952. However, it is noted that their criteria were rigid and conformed essentially to those of Drake et al.: (1) Low serum calcium; (2) high serum inorganic phosphorus (greater than 7.0 mg. percent) (Steinberg and Waldron); (3) absence of renal insufficiency which may produce hyperphosphatemia; (4) normal bones by roentgenography (to exclude infantile rickets and adult osteomalacia); (5) chronic tetany.

Idiopathic hypoparathyroidism is replete with signs of neuro-ophthalmologic interest. Reproductions of Tables VI, VII, and VIII from Steinberg and Waldron are valuable in themselves and serve to eliminate much description here.

The signs of idiopathic hypoparathyroidism selected for comment are: conjunctivitis, keratitis and blepharospasm, cataracts, papilledema, calcification of the basal ganglia.

Conjunctivitis, keratitis, and blepharospasm

Included in Table 2 (Table VII—Steinberg and Waldron) there is a single case characterized by photophobia and blepharospasm. In six cases that have come under my observation, photophobia and blepharospasm have represented major reasons for disability in two instances. These two cases are briefly summarized:

J. J. (150390), reported by Emerson, Howard, and Walsh, was seen as a young boy. Photophobia and blepharospasm were principal com-

TABLE 2

IDIOPATHIC HYPOPARATHYROIDISM

Table VI (Steinberg and Waldron) Pediatric and Adult Distribution

Group	*Male*	*Female*	*Total*
Pediatric (16 years or less at onset of symptomatology)	15	19	34
Adult	9	6	15

Table VII (Steinberg and Waldron) Tabulation and Incidence of Symptoms

Symptoms	*Number of Patients*	*Percentages*
Tetany	39	7
Convulsions	26	52
With tetany and/or "tetanic equivalents"	21	
Without tetany or "tetanic equivalents"	5	
Tetanic equivalents (paresthesias, numbness, cramps and/or laryngeal stridor) without carpopedal spasm	5	10
Gastrointestinal disturbances	4	8
Constipation	2	
Nausea and vomiting	1	
Unspecified	1	
Psychosis	2	4
Nervousness	2	4
Photophobia and blepharospasm based on 50 cases	1	2

Table VIII (Steinberg and Waldron) Tabulation and Incidence of Signs

Signs	*Number of Patients*	*Percentages*
Cataracts	26	50.0
Poor dentition	19	36.5
Skin, hair and/or hair changes	13	25.0
Papilledema	7	13.5
Fungus infections	7	13.5
Mental retardation	4	7.7
Generalized physical retardation	3	5.8
Chronic conjunctivitis	2	3.8
Syndactylism	1	1.9
Polydactylism	1	1.9

plaints. There were classical evidences of hypoparathyroidism as regards tetany, blood-serum calcium, and phosphorus, absence of renal involvement, and changes in the skin, teeth, and hair.

Examination of the eyes showed low-grade conjuncitval irritation, normal corneas after fluorescein stain, small opacities in the cortices of the lenses, and intense photophobia and blepharospasm.

It was recorded in the history that a previous examiner had said phlyctenular conjunctivitis had been present.

Treatment with A. T. 10, later with vitamin D, produced spectacular improvement.

A. O'M. (34883), a white girl, was seen through the courtesy of Dr. Lawson Wilkins. The child was first studied when she was four years of age. The history was that, at one year of age, there was development of a papular rash on the face, and with it there was development of blepharospasm. Somewhat later the child developed diarrhea. The blepharospasm continued.

At four years, nine months it was found that fluorescein stain was taken up by both corneas. Also at this time evidences of tetany were recognized during physical examination. Serum calcium was 4.0 mg. percent; inorganic phosphorus 10.3 mg. percent; phosphatase 12.1 units. There was no renal impairment.

Percomorph oil was administered without beneficial response. With A. T. 10 the serum calcium was increased and the phosphorus was reduced. There was great improvement as regards the photophobia. Parathormone was found to influence favorably the low calcium; high phosphorus of the blood serum eliminating pseudohypoparathyroidism.

During several years co-operation between parents and attending physicians was not good and, as a result, during the major part of this time the tetany was not thoroughly controlled. Blepharospasm became a major complaint and the child was unable to get about by herself because of inability to open the eyes.

Approximately six months ago, when the child was 11 years of age, Dr. Wilkins succeeded in hospitalizing her. Thorough studies which are not detailed here satisfied him that idiopathic hypoparathyroidism was the correct diagnosis.

Before the tetany was controlled the blepharospasm was intense and the eyelids were closed constantly. The conjunctivas were red and injected. Both corneas were semi-opaque so that it was difficult to identify the pupils. There was massive superficial vascularization of both corneas and the fundi could not be seen. The visual acuity was greatly reduced. It was estimated that it amounted to perception of hand movements.

It was again shown that vitamin D in doses of 100,000 units per day produced a dramatic response as regards relief from the blepharospasm.

Now some six months after I first examined her, the eyes are almost white. The corneal opacities persist and the vascularization is unchanged but the visual acuity is approximately 10/200 for each eye. The blepharospasm has been relieved and even slit-lamp examination can be fairly successfully undertaken without assistance. Lens opacities are present but the corneal changes are in largest part responsible for the low visual acuity.

Summary. In two cases of hypoparathyroidism blepharospasm has been relieved by the administration of A. T. 10 and control of tetany with vitamin D is continuing to be successful, in the more severe case of these reported two. In both there was early skin involvement.

Sutphin et al., in reporting five cases of idiopathic hypoparathyroidism, included the case of a 12-year-old girl who also exhibited evidences of moniliasis and Addison's disease. Ectodermal changes were present. Phlyctenules were present at the limbus of each cornea. The conjunctivas were injected and there was photophobia and blepharospasm.

Leonard described a case of a nine-year-old girl with Addison's disease and idiopathic hypoparathyroidism who exhibited bilateral papilledema and showed in roentgenograms bilateral cerebral calcification. Five months prior to death this child developed what was diagnosed as phlyctenular conjunctivitis with photophobia, active corneal ulcers, and blepharospasm.

Corneal clouding was bilateral in a case described by Eaton, Camp, and Love; a corneal ulcer developed in one eye; further details were not available in their paper.

The etiology of conjunctivitis, keratitis, and blepharospasm in idiopathic hypoparathyroidism is unknown. The incidence is not known with any certainty. Several interesting features characterize the cases mentioned in this section.

Skin involvement or abnormalities of the hair, teeth, or nails was present in all five cases. There were two of the five cases in which Addison's disease was associated with the parathyroid disorder. Widespread tuberculosis was present in one of the cases of Addison's disease. In the other case at post mortem, no tuberculosis was identified but there was atrophy of cortical adrenal tissue.

In three of the five cases a diagnosis of phlyctenular conjunctivitis was made at some time during the course of the affection.

It would seem obvious that the presence of phlyctenular conjunctivitis should always suggest estimation of serum calcium and phosphorus. Furthermore, persistent conjunctivitis, keratitis, and blepharospasm should invariably call for a similar examination of the blood.

Cataracts in hypoparathyroidism

Lens opacities develop in a majority of individuals who suffer from hypoparathyroidism whether it is of the idiopathic or acquired variety according to our experience and that of others. In a minority of cases the cataracts have progressed to a degree that indicates the necessity for extraction. In a large majority of cases slitlamp evidence consists of dotlike opacities situated rather well away from the capsule.

Barr et al. have stated that administration of A. T. 10 will stop the progress of lens opacities in hypoparathyroidism. Vitamin D functions similarly as we have observed in a single instance (J. S. A-70998). When cataract extraction is undertaken one can look forward with confidence to a satisfactory result.

As regards lens changes in hypoparathyroidism, it is unlikely there is any difference between those seen in the idiopathic and the acquired variety. Duke-Elder has stated that the mineral content of hypoparathyroid lenses is not higher than that of senile cataracts. (As regards senile cataracts, Salit classifies cataracts on the calcium content of the lenses; up to 10 mg. percent, net weight, incipient; 10 to 35 mg. percent, immature; over 35 mg. percent, mature cataract.)

Particular mention is made of Clark's work in regard to permeability of the lens capsule and the calcium content of the lens in pigs' eyes since it would seem to bear on the cataracts of hypoparathyroidism. Working with ultraviolet light and solutions containing calcium she found that calcium would pass through the lens capsule. If, however, parathyroid extract was added to the solutions no calcium reached the lens substance.

Earlier work of Kirby seemed to show that the lens requires calcium; he found that living epithelial cells in vitro continue to live in solutions with a high calcium content but if calcium is removed from the solutions "toxic" responses develop in the epithelial cells.

Possible relationships between parathyroid deficiency and congenital cataracts may be mentioned here. Infantile tetany (Bakwin), which rather often is recovered from spontaneously or through administration of calcium, may be responsible for the development of lamellar cataracts which are identified months or years later.

Goldmann's experiments are of importance in this regard; he found that in experimental animals during tetany subcapsular opacities developed in the lenses; by obtaining periods of tetany and periods of freedom from it he produced concentric rings of opacities in the lenses of the animals.

Papilledema and optic neuritis in hypoparathyroidism

Occasionally, papilledema develops in cases of idiopathic or acquired hypoparathyroidism. Probably optic neuritis occurs in individuals suffering from hypoparathyroidism but it has been reported infrequently and may have been fortuitous.

Albrecht collected eight cases and added one of his own all exemplifying papilledema; autopsy in one case revealed extreme swelling of the brain. Martell also found profound edema in such a case.

Barr, MacBryde, and Sanders described two cases: their first case seemed to exemplify optic neuritis; their second case was one of idiopathic hypoparathyroidism with a history of attacks of loss of consciousness, evidences of latent tetany, a serum calcium of 5.9 to 6.5 mg. percent, phosphorus 6.5 to 7.3 mg. percent, anterior subcapsular cataracts, and three diopters of papilledema in each eye. Treatment with A. T. 10 produced immediate improvement as regards the tetany and a rapid disappearance of the papilledema.

Lyle described the case of a 52-year-old woman who, four years earlier, had been subjected to thyroidectomy. Following the operation she showed evidences of hypoparathyroidism and developed cataracts which were extracted approximately four years after thyroidectomy. Although attention had been given to the hypocalcemia just before the second cataract was extracted, nine days after operation the patient developed convulsions. At this time serum calcium was 6.5 mg. percent, phosphorus 2.35 mg. percent. There was bilateral papilledema without hemorrhages. Improvement followed institution of treatment with A. T. 10.

Hurxthal recorded the case of a 26-year-old woman who had had a thyroidectomy seven years before his examinations. Air studies gave normal findings. A subtemporal decompression was performed. Papilledema persisted for almost a year when it largely disappeared only to recur with recurrence of intracranial pressure. During the period the papilledema was being observed, the convulsions, which with headaches were responsible for her seeking aid, were controlled and A. T. 10. was more satisfactory than other therapy.

Shelling and Goodman remarked on the presence of choked discs in a 32-year-old woman who had been subjected to thyroidectomy six years earlier. At the time this patient developed convulsions she exhibited choked discs.

Leonard's case was one of idiopathic hypoparathyroidism and Addison's disease and with three diopters of papilledema; recovery as regards the general symptomatology and the papilledema followed therapy directed to the hypoparathyroidism.

Sutphin et al. observed bilateral papilledema in a 12-year-old boy who was admitted with convulsions, drowsiness, spinal fluid pressure 250 mm. water. Ventriculograms and encephalograms gave normal findings. Treatment with A. T. 10 produced rapid improvement in the general symptomatology. The papilledema disappeared completely within a matter of months.

As regards optic neuritis, Barr, MacBryde, and Sanders remarked on their

Case 1 having been diagnosed as optic neuritis. Lillie described optic neuritis in hypoparathyroidism.

Papilledema in hypoparathyroidism would appear to be characterized by:

1. Some signs of parathyroid deficiency may be present for years before the onset of severe symptoms such as convulsions and loss of consciousness.

2. Pronounced hypocalcemia is invariably present when papilledema is identified.

3. The papilledema is low-grade in most instances, a maximum of three diopters in several case histories studied; also in several instances it was remarked that hemorrhages were absent.

4. Spinal fluid pressure, not mentioned in some records, was not unduly high when remarked upon.

5. The spinal fluid when examined was not reported as showing an abnormal protein content, also it had normal calcium and phosphorus content.

The etiology of the papilledema in these cases is not known; it is reasonably established that cerebral edema is present but the explanation for the cerebral edema is not at hand. It is interesting to speculate as regards a difference in calcium content on the two sides of the hemato-encephalic barrier being responsible for the edema, but this is not established.

Finally, attention is directed to the concurrent Addison's disease in some cases of hypoparathyroidism. Recently papilledema has been observed in a case of Addison's disease; the papilledema disappeared spontaneously within three months.

The possibility that hemoconcentration and low blood pressure favored the development of dural sinus thrombosis was offered as a reasonable explanation for papilledema (Walsh).

Papilledema in an individual exhibiting convulsions or with a history of convulsions should always suggest the possibility of hypoparathyroidism just as surely as do convulsions in the absence of papilledema. This generalization holds even when general physical examinations fail to reveal evidences of tetany.

At the time of writing we have under treatment a woman whose husband had been advised she should be committed to a mental hospital. She had been subjected to thyroidectomy four months ago. On admission she was semicomatose. General physical examination revealed sluggish or absent deep reflexes, and absence of Chvostek's and Trousseau's signs. Serum calcium was 3.5 mg. percent, inorganic phosphorus, 2.7 mg. percent. With therapy (intravenous calcium and vitamin D) recovery ensued and evidences of tetany became manifest. No lens opacities were visible on her discharge from hospital (M.P. 521179).

It would seem reasonable that, in cases of unexplained papilledema, serum calcium estimation might point to the diagnosis in occasional instances, since latent tetany is easily overlooked or may be temporarily absent.

Symmetric calcification of the basal ganglia

Bilateral cerebral calcification is a frequent autopsy finding and it occurs in association with a variety of conditions: amaurotic idiocy, encephalitis, and lead intoxication to mention only a few (see Eaton, Camp, and Love).

Microscopic concretions are found frequently in the basal ganglia when there is no evidence of any disease process. This may explain the predilection to sizeable calcifications in this region. Undoubtedly calcifications are too minute to be shown on X-ray films in many instances.

Roentgenologically visible calcifications in the basal ganglia have been present in several cases of idiopathic hypoparathyroidism. Camp reported on 12 such cases. Calcifications were observed in a parathyroprivic case by Siglin et al. who suggested that the case supported the idea that the hypoparathyroidism was directly responsible for the calcification. However, as Foley stated, it is remark-

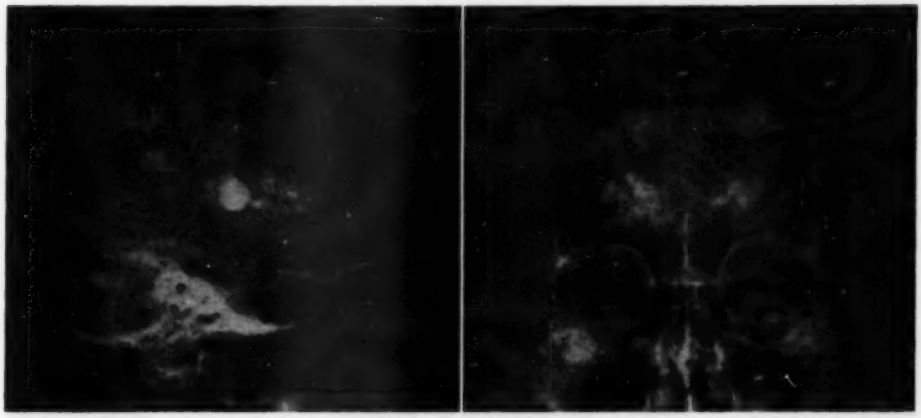

Fig. 4 (Walsh and Murray). Roentgenograms showing calcification of the cerebral basal ganglia and cerebellum (R. B. R.).

able that such calcifications are not observed more frequently in such cases as Siglin et al. reported since surgical removal of the parathyroids is of common occurrence and a frequent cause for hypoparathyrodism.

Pseudohypoparathyroidism, which may be familial, has been associated with calcification of the basal ganglia and with metastatic calcinosis (Alexander and Tucker).

Foley described cerebral calcification in the caudate, lenticular, and dentate nuclei of a woman, aged 60 years, with intellectual impairment, spasticity, and ataxia; her two daughters had similar calcifications but no neurologic symptoms. The familial occurrence of cerebral calcifications in the absence of hypoparathyroidism or other disorder was described thoroughly by Foley who reviewed the literature. The condition is of infrequent occurrence.

Symmetrical calcification of the basal ganglia has not been described as characteristic of toxoplasmosis. However, Camp has suggested toxoplasmosis as a probable diagnosis in twin boys in whom such calcification was present.

I have examined recently these boys some 10 years after Camp's studies were made. They present an interesting symptom-complex which is incompletely summarized below. It is highly doubtful that toxoplasmosis is responsible for the disease picture. I am unable to make a diagnosis. The cases are mentioned here because of their intrinsic interest.

Two brothers, Robert R. (612151) and Daniel R. (612680), identical twins, 18 years of age, at the time of my examination exhibited multiple defects: canities, cataracts, genital hypoplasia, calcification of the basal ganglia (and cerebellum, Robert), a yellow tinge to the skin and a few cafe au lait spots. Robert had epileptiform seizures and unilateral ataxia; Daniel exhibited cogwheel rigidity minimally.

Robert's vision was: R.E., Light perception; L.E., no light perception. Daniel's vision was: R.E., hand movements; L.E. 20/20.

No fundus details could be made out in Robert's eyes. In Daniel's right eye there were dark heavy vitreous opacities and elevated gray areas in the posterior fundus (difficult to see). The left fundus was essentially normal. Slitlamp examination of Robert's eyes and Daniel's right eye suggested earlier inflammation.

These boys weighed just less than four pounds at birth. Except for a tremor of the right hand and leg, occasionally noted in Robert, development was considered as normal until they were nine years of age. Then it was discovered that there was progressive loss of vision. They were found to have lens changes. The fundi, then visible, presented a picture that might be angiomastosis or Coates' disease. Then calcification of the basal ganglia was shown to exist and the diagnosis of toxoplasmosis was suggested.

Laboratory tests made on these boys when they were 18 years of age failed to establish a diagnosis of toxoplasmosis, or to point to any other diagnosis. (figs. 4 and 5).

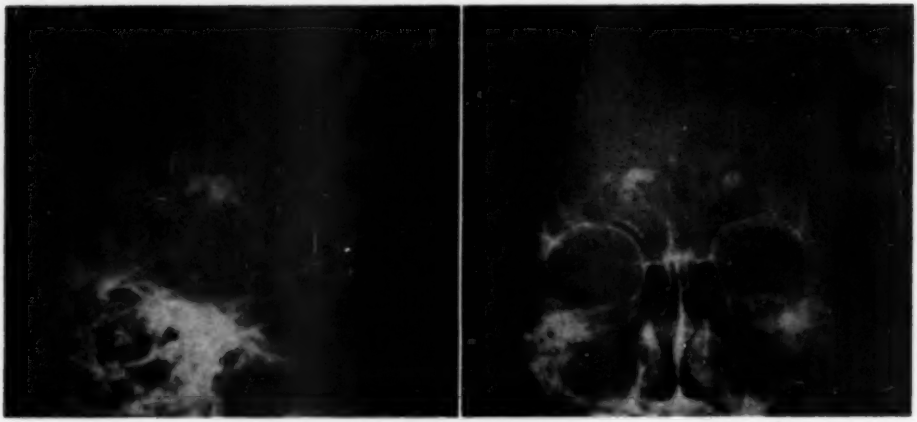

Fig. 5 (Walsh and Murray). Roentgenograms of the twin brother (D. R. R.).

Through the courtesy of Dr. John A. Wagner* we have seen sections of the brain from an unusual case of hypoparathyrodism. The case merits attention for two reasons: the 40-year history and the extensiveness of the cerebral calcification. The case history is incompletely summarized.

A 53-year-old white woman had been subjected to thyroidectomy when she was 13 years old. Her last admission to hospital was March 10, 1949; she died six days later.

When the patient was 30 years of age she commenced having convulsions which, after some delay, were recognized as being associated with chronic hypocalcemia. Treatments were directed toward this almost continuously until the time of her death.

On her last admission she was unco-operative, stared into space, and had repeated convulsions. The pupils were wide and responded to light and convergence-accommodation. The fundi seemed normal save for low-grade retinal arteriosclerosis.

Blood studies showed hypocalcemia on several occasions (a typical example: calcium 5.1 mg. percent, phosphorus 7.6 mg. percent). Sulkowitch test for urinary calcium, negative. Electrocardiograph showed prolonged Q-T, the interval being interpreted as indicative of hypocalcemia.

X-rays studies of the skull showed evidence of a craniotomy wound on the right side. There was cerebral calcification beneath the parietal pole and area. There was no abnormality of the long bones. There was no response to therapy and death occurred, seemingly as the result of shock.

Autopsy. Examination of the gross brain showed the ventricles to be of normal size. Throughout the basal nuclear structures and in the internal capsules there were deposits of calcium. For the most part the calcification was of fine granular type showing on the cut surfaces as a slightly raised punctate granularity. Such changes extended into the centrum semiovale on either side. The calcium depositions extended throughout the greater portion of the cerebral hemispheres and the dentate nucleus of the cerebellum.

Microscopic examinations showed an essentially normal cortex except for a few small foci of degeneration in the third and fourth layers. In some areas of cortex there was extreme calcification of capillaries. Occasionally neuronal cells were replaced by calcium depositions and such changes in several cells produced a satellite appearance. In general, the calcific changes were predominantly in the capillaries. In the white matter calcification of the arterioles was seen throughout, and much of the media was replaced by calcific material.

Comment. In this case the cerebral calcifications are widespread with the basal ganglia and cerebellum particularly involved. The vascular changes pointed out in the microscropic examination are of particular interest.

IV. Local disturbances in calcium metabolism

Local calcifications

It is generally well known among pathologists that there is a tendency toward the deposition of calcium salts in inflamed tissues

* Note. This patient was studied in the Department of Medicine and Pathology, Lutheran Hospital of Maryland, Baltimore. The material above has been written from notes supplied by Dr. Wagner.

and in areas of fatty or hyaline degeneration. Such calcifications, sometimes termed "dystrophic," is dependent upon mechanisms which are not understood. Whether generalized or widespread involvements such as myositis ossificans, scleroderma, and calcinosis (which is widespread) should be included in this category is debatable. In any event discrete and localized calcifications are not associated with changes in the blood serum as regards calcium or phosphorus.

Obviously, band keratitis secondary to ocular inflammation is a local calcification as also are calcification of the choroid (almost always amounting to true bone formation) and calcification of the lens. Hyaline bodies of the optic disc and corpora amylacea in the optic nerve sheaths are prone to become calcified.

Other local calcifications which are demonstrable in roentgenograms are of interest to ophthalmologists. These are situated within

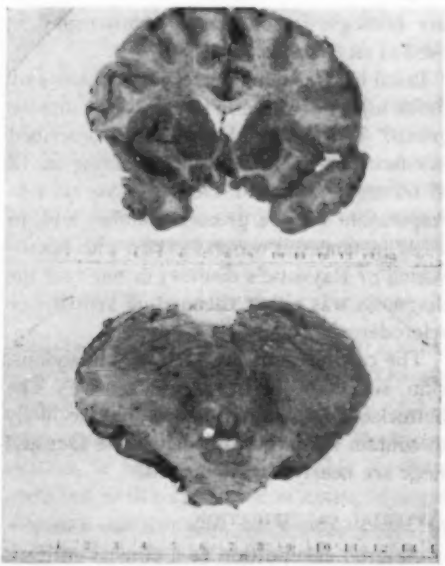

Fig. 6 (Walsh and Murray). Sections of brain of a 53-year-old woman who had suffered from hypoparathyroidism for at least 23 years. Calcium deposits show up as black spots (courtesy Dr. Wagner).

Fig. 7 (Walsh and Murray). Calcium in retinoblastoma.

the bones of the skull or within the cranial activity and here further mention is made only of calcifications within the cranium. These may be grouped as follows:

1. Physiologic calcification is exemplified by calcification of the pineal gland, the petrosphenoid ligaments, and in the choroidal plexuses of the lateral ventricles.

2. Calcification with neoplasms: this occurs in some cases of intraocular neoplasm-retinoblastoma (fig. 7). In children particularly, characteristically placed areas of calcification suggest the possibility of craniopharyngioma; areas of calcification may be associated with meningiomas, gliomas, or dermoid cysts. Angiomas are frequently "spotted" because of the presence of areas of calcification. In the Sturge-Weber syndrome the appearance of the calcified areas is highly characteristic (figs. 8 and 9).

3. Calcification may be associated with hemorrhage and has been observed in subdural hematomas of long standing. Hemorrhages into the basal ganglia may be responsible in rare instances for symmetrical calcification in this region. Cerebral hemorrhage or thrombosis with later infarction of the cerebrum may rarely explain cerebral calcification.

4. Inflammations of the meninges and cerebrum may precede the development of calcification.

5. Parasitic infestations are frequently characterized by the development of cerebral calcification. A classical example is toxoplas-

Fig. 8 (Walsh and Murray). Sturge-Weber's syndrome. Classical calcification of cerebrum (courtesy Dr. George Smith).

mosis (fig. 10). Trichiniasis and echinococcosis also may be etiologic.

6. In many instances cerebral calcification is present when it is impossible to visualize any etiologic factor; facial hemiatrophy is cited as an example.

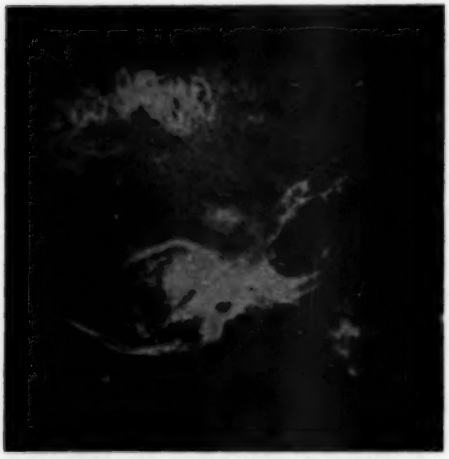

Fig. 9 (Walsh and Murray). Extensive calcification in case of bilateral Sturge-Weber syndrome (courtesy Dr. George Smith).

Local calcifications present on the basis of tissue injury are seen in the "collagen disease group." Wheeler and his associates described calcifications of soft tissue occurring in 12 of 66 cases examined. Dermatomyositis was responsible for the greatest number and, in a few instances, it was associated with scleroderma or Raynaud's disease; in one case the diagnosis was either rheumatoid arthritis or scleroderma.

The calcifications are seen in the tendons, skin, subcutaneous tissue, and muscles. The buttocks, thighs, arms, and trunk are likely to contain the largest deposits. The face and neck are nearly always spared.

Arterial calcification

Arterial calcification as a clinical entity is not elaborated in this paper. It has already been noted that calcification is prone to occur in elastic tissue. It follows that calcification is associated frequently with atherosclerosis.

However, calcification does not always confine itself to the intima and the media and adventitia are involved in some instances.

It is generally known that atherosclerosis selectively involves the aorta and its branches and these include the internal carotid and ophthalmic arteries. Friedenwald has shown that atheromatous plaques may be found in the central retinal artery and that they occur most frequently in its neural portion but may be found in the intraocular portion. Such plaques are liable to calcification particularly in the larger vessels but intraocular calcification of retinal arteries occurs rarely.

Calcification of the internal carotid artery has received much attention in the recent past; loss of the nasal field has been attributed to such arterial calcification. It is now fairly generally agreed that calcification of the carotid artery is not established as a cause for "soft glaucoma." It seems probable that interruption to nutrient vessels to the nerve through sclerotic changes in them is responsible for the loss of field in some cases.

It has been shown by Alpers et al. that the retinal ophthalmoscopic picture of atherosclerosis does not justify conclusions regarding the state of the cerebral vessels beyond the probability that when there is present an atherosclerosis of the retinal vessels the basilar artery will exhibit arteriosclerosis. These investigators have shown that retinal and cerebral arteriosclerosis are independent involvements.

Angioid streaks, pseudoxanthoma elasticum, and peripheral arteriosclerosis

In the light of arterial calcification being dependent upon elastic tissue and its degeneration, it has seemed reasonable to draw attention to this syndrome as exemplifying a selective involvement of elastic tissue in various body structures—Bruch's membrane, the skin, and the arteries of the extremities. Klien's paper has been used extensively in the preparation of this section.

Klien credited Groenblad as being first to suggest that angioid streaks and pseudoxanthoma elasticum might represent a syndrome of selective degeneration of elastic tissue. She noted that Finnerud and Nomland showed that deposition of calcium phosphate occurred in the skin of individuals exhibiting pseudoxanthoma elasticum. Earlier, with Nomland, Klien had suggested that fibrils of elastic tissue in the subpapillary and midcutis layers of the skin probably contained calcium.

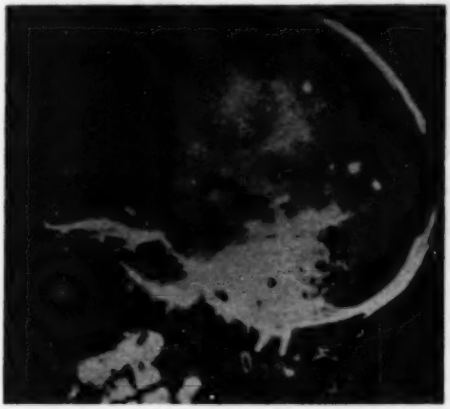

Fig. 10 (Walsh and Murray). Calcification in toxoplasmosis.

In her Case 6, Klien showed that Bruch's membrane, particularly in its outer parts, contained calcium as was proven by the use of von Kossa reagent. In this case, it was shown that iron salts were present in addition to salts of calcium and Klien thought they were deposited later than the calcium. The calcification of Bruch's membrane was spotty during the early stages and at a time when the degeneration of the membrane was also spotty. She concluded:

"The clinical picture of angioid streaks represents not only the visible ruptures in Bruch's membrane, but also multiform end results of irritation of pigment epithelium and choriocapillaris by the sharp calcified edges and fragments of the broken membrane. Calcification, therefore, even if secondary to the elastic degeneration in the pathologic process, is essential in production of all except, perhaps, the earliest manifestations of the disease."

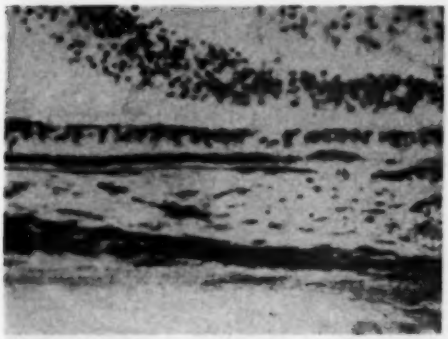

Fig. 11 (Walsh and Murray). Spotty black stain of Bruch's membrane with Kossa test for calcium (×400). Thick cuticular layer under pigment epithelium (Figure 7, Klien).

Scheie and Freeman, in 1946, drew attention to the occurrence of sclerosis of the peripheral arteries of the extremities in individuals exhibiting angioid streaks and pseudoxanthoma elasticum, namely, degeneration of elastic tissue (fig. 11).

Although osteitis deformans (Paget's disease) is not considered here in any detail it is of interest that angioid streaks are occasionally observed in association with Paget's disease. Klien is authority that Paget's disease and pseudoxanthoma elasticum have not been observed in the same individual.

It is well known that in Paget's disease there are bony changes which are relatively paralleled by arteriosclerosis. Furthermore, in Paget's disease the alkaline phosphatase is usually elevated but the serum calcium and phosphorus are within normal limits in a great majority of cases.

Metastatic calcification has been observed as part of Paget's disease by Wells and Holley who noted that in their case it might have been due to ingestion of large amounts of vitamin D. They found calcification of the gastric mucosa in their case.

I have observed angioid streaks and pseudoxanthoma elasticum in two patients who had suffered from repeated gastric hemorrhages; no evidences of Paget's disease were recognized in either case (T. 582989).

Revell and Carey have described calcification of arteries in the extremities, vascular hypertension, and gastrointestinal bleeding as evidences of dissemination of pseudoxanthoma elasticum.

Summary and Conclusions

An effort has been made to correlate abnormalities concerning calcium with selected groups of cases of ophthalmologic interest. Basic facts regarding calcium have been restated.

1. Changes in the cornea, conjunctiva, and sclera occur in association with hypercalcemia. Such changes have been redescribed. The corneal changes are more readily recognized than those in the conjunctiva and must be considered as more specific.

2. Hypocalcemia is in some instances observed in patients exhibiting blepharospasm, conjunctivitis, and keratitis; several such cases are described in the section on idiopathic hypoparathyroidism. In such cases other ectodermal defects have invariably been present. It is suggested here that such external involvements should suggest the possibility of idiopathic hypoparathyroidism to the examiner. Phlyctenular conjunctivitis was diagnosed at some time during the course of the affection in three of five of these cases. It would seem to follow that the presence of phlyctenular disease calls for a serum calcium estimation.

3. As regards the lens in hypocalcemia, it is remarkable that the calcium content is likely to be greatly increased. The mechanisms by which such increased calcium content occurs are not thoroughly understood. It is noteworthy that with hypercalcemia there are not any reported lens changes.

4. Angioid streaks and pseudoxanthoma elasticum (Groenblad's syndrome) and arteriosclerosis of the extremities represent a selective involvement of elastic tissue. Mention is made of the relationship which may exist between Paget's disease and Groenblad's syndrome.

5. Papilledema is observed in some cases

of hypoparathyroidism. Its pathogenesis is not clear. In many such cases, years have elapsed between the first symptoms of hypoparathyroidism and the development of papilledema. When convulsions are present an estimation of blood serum calcium may point to the diagnosis and serve to eleminate air injection, anteriograms, or craniotomy. Indeed, in unexplained papilledema just as in unexplained convulsions, an estimation of serum calcium may be useful. Latent tetany is overlooked frequently.

6. Cerebral calcification has received attention as a local (dystrophic) process. Calcification which has a particular tendency to involve the basal ganglia is of fairly frequent occurrence in hypoparathyroidism and occurs also in families when there is no available evidence of a metabolic defect. It has been described here for its intrinsic interest and because of the complex symptomatology seen in twin boys who exhibited in addition to cerebral calcification extensive changes in the optic fundi; the chronology of the signs made it seem unlikely that toxoplasmosis could account for the entire picture. We have been unable to make a diagnosis in what may be a recognizable symptom-complex.

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Ophthalmic Miniature

For a long time, he had been deprived of the use of one of his eyes, which was blinded by cataract, how long he could not tell with accuracy,—for the discovery that it was useless, and that he saw only with the other, was made by accident, and much to his surprise—but it must have been more than twenty years. Opening it one morning while the right eye was buried in the pillow, he found himself unable to perceive any objects about him. For many years, however, he saw well enough for common purposes with the other; but, more recently, even that one had caused him so much trouble that he lived in fear of total blindness. Early in 1853, cataract appeared in that eye also, and was making such rapid progress that in a few weeks all vision was lost. Under these circumstances, he resolved to submit to an operation on the one which had been so long obscured. It was successfully performed by Dr. H. W. Williams, of Boston, the cataract being broken up in the month of March. Some time was necessary for the complete absorption of the fragments; but, in less than three months, the pupil had become entirely clear and, by the aid of cataract-glasses, he could not only see large objects as well as ever, but could read the newspapers, and even the fine print in the column of ship-news. His sight was at times rendered feeble afterwards by the general debility of his system, and he never recovered the power of reading and writing with entire ease; but to do both in some degree was an advantage, in comparison with total loss of sight, that could hardly be appreciated, particularly as it enabled him still to manage his own affairs, which he always wanted to do, and did to his last day—even keeping his books with his own hand, except for a few months of his last year, when the entries were made from his dictation.

Thomas G. Cary,

*Memoir, Thomas Handasyde Perkins,** 1856, p. 259.

* The first of that name, son of James Perkins, was born in 1764 and died in 1854. He was the founder of the Perkins Institute for the Blind.

THE OCULAR EFFECTS OF METHYL ALCOHOL POISONING:*

REPORT OF A CATASTROPHE INVOLVING 320 PERSONS

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AND

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On October 20, 1951, a large quantity of bootleg whiskey containing 35 percent methyl alcohol and 15 percent ethyl alcohol was distributed in Atlanta, Georgia. During the succeeding seven days, 320 persons sought emergency medical care at Grady Memorial Hospital because of symptoms attributable to the ingestion of this mixture. Twenty-four persons died before or shortly after reaching the hospital and 13 died on the medical wards, making a total of 37 deaths. A general report of these cases has been published elsewhere.[5]

Ophthalmic observations were made by one of us (C.D.B.) on a large proportion of the patients immediately upon their acceptance to the hospital emergency room. Data were recorded concerning the amount of contaminated whiskey consumed and the date thereof, onset and character of symptoms, the vision, pupillary reaction to light, visual fields (confrontation method), and ophthalmoscopic appearance of the fundi.

Every patient admitted to a hospital bed was examined daily until the time of his discharge and then at gradually increasing intervals in the eye clinic. Measurements of visual acuity and central visual fields and observations of the ocular fundi were made at each visit during the eight months' follow-up period. Patients who were never admitted to the hospital were asked to report to the eye clinic for examination; 123 patients returned for re-examination.

SYMPTOMS

The symptoms of acute methyl alcohol poisoning usually developed 18 to 48 hours after ingestion, but in a few instances transitory symptoms were noted within one hour. The patients complained of visual disturbances, weakness, abdominal pain, nausea and vomiting, headache, dizziness, and shortness of breath. Some patients had consumed as little as 90 cc. of the alcoholic mixture and others had drunk as much as 750 cc. Within this range there was no close correlation between the severity of symptoms and the quantity of methyl alcohol consumed.

The initial visual symptoms ranged from spots before the eyes to complete blindness. Many patients complained of whitish or grayish misty vision. The visual acuity was either normal or markedly reduced. The patients with severe visual loss could count fingers only in the midperiphery of their visual field. Three patients who were conscious had no light perception in either eye.

Characteristically, the initially reduced vision showed an early recovery which was in some cases only transitory. Many patients experienced full return of vision during the first hour of treatment by intravenous alkaline fluids. All patients who regained and retained normal visual acuity did so within six days after treatment was begun.

If the vision did not return to normal in six days, it invariably dropped again to a very low level. Only three patients, all nearly or completely blind at the time of

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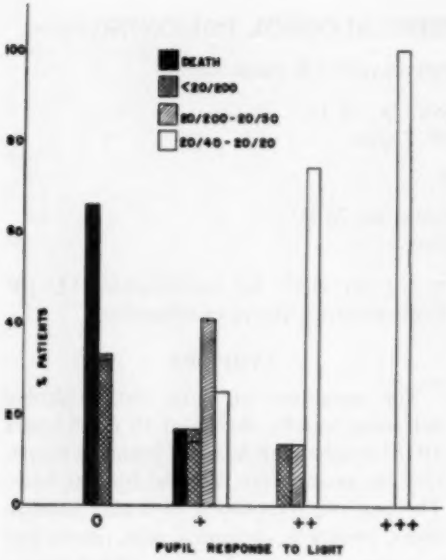

Fig. 1 (Benton and Calhoun). Correlation of pupillary response to light with fatality and final vision (+++ equals normal reaction).

hospital admission, failed to exhibit any permanent or temporary improvement in vision.

In the acute phase of poisoning, diminution of the pupillary reaction to light occurred in all patients with impaired vision and in many patients with normal vision. The degree of impairment of the pupillary light reflex proved to be of considerable prognostic value. Patients with dilated, fixed pupils usually died; if they recovered, they always had severe visual damage. Lesser degrees of impairment in the pupillary response to light were indicative of a more favorable outcome and no patient with normally reacting pupils had permanent visual loss. These findings are illustrated in Figure 1.

The ophthalmoscopically visible fundus changes were always identical in type and sequence of appearance and varied only in intensity from one patient to the next. A hyperemia of the optic disc (color plate, fig. a) could be seen at the time symptoms of visual loss developed. The reddish color of the papilla persisted from one to seven days.

From six to 24 hours after the hyperemia became visible, there developed a whitish striated edema of the disc margins and adjacent retina. Edema was most extensive along the course of the major retinal vessels, and it persisted for 10 to 60 days. The peripapillary retinal edema (color plate, figs. b and c) appeared to be chiefly in the nerve fiber layer. Engorgement of the retinal veins usually accompanied the retinal edema and followed a similar course.

These characteristic fundus changes were seen in 87 percent of patients with initial visual loss and in every patient who later showed permanent impairment of vision.

Mild or moderate retinal edema was followed by complete recovery of vision in some cases, blindness in others. It was found, however, that all patients who had severe retinal edema and most patients with moderate retinal edema had some degree of permanent visual loss.

In patients with severe ocular damage, an atrophy of the optic disc became visible in 30 to 60 days. Usually the optic nerve atrophy (color plate, fig. d) was identical in appearance to so-called primary optic atrophy, although occasionally a cupping of the nerve-head that simulated glaucoma was seen.

In five patients there was a striking difference between the visual acuity of the two eyes, even though the ophthalmoscopic changes were always bilaterally equal. In four of the five such cases, the left eye was the most severely impaired.

The shape of the scotomas offered the explanation of this difference in visual acuity. In the central field of the better eye, the scotoma extended from the blindspot like an open mouth preparing to engulf the fixation area. In the opposite eye, a complete centrocecal scotoma obliterated the area of critical vision. After four to six months, the visual acuity in the better eye began to decline and approach the level of the other eye.

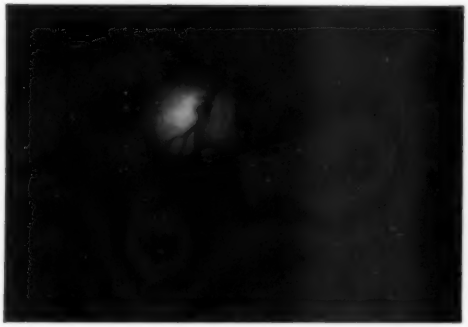

FIG. A

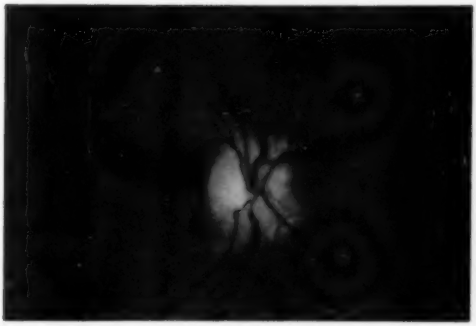

FIG. B

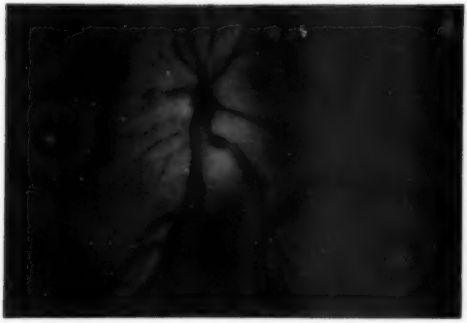

FIG. C

FIG. D

FIGS. A TO D (BENTON AND CALHOUN). ACUTE METHYL ALCOHOL POISONING.
(FIG. A) STAGE I. HYPEREMIA OF THE OPTIC DISC.
(FIG. B) STAGE II-A. MILD PERIPAPILLARY RETINAL EDEMA.
(FIG. C) STAGE II-B. SEVERE PERIPAPILLARY RETINAL EDEMA.
(FIG. D) STAGE III. OPTIC ATROPHY.

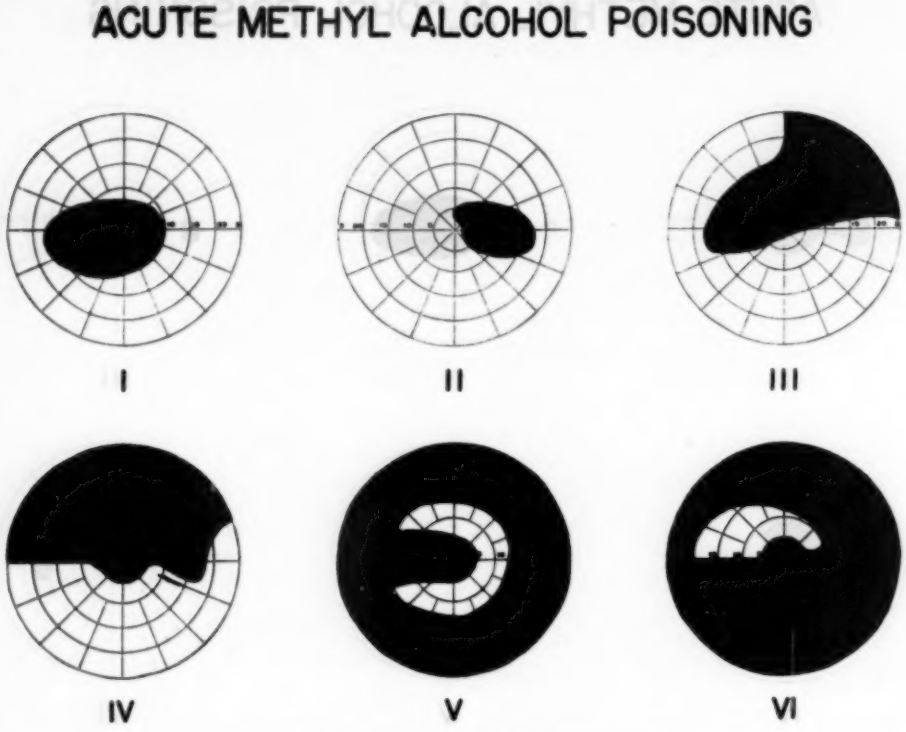

Fig. 3 (Benton and Calhoun). Acute methyl alcohol poisoning. Late central field defects (5/1,000 white test object).

combining capacity of the blood. The blood CO_2 was measured on every patient immediately upon his acceptance into the emergency clinic. The degree of acidosis proved to be an invaluable guide to diagnosis, therapy, and prognosis. This point has been repeatedly stressed by Roe and others since 1943.[4, 7, 14–17]

The blood CO_2 was normal (above 26 milliequivalents per liter, or 55 volumes percent) in 188 patients. Not one patient in this group had genuine symptoms or signs of methanol poisoning and undoubtedly many of these patients drank none of the contaminated whiskey. No treatment was given any patient who had a normal blood CO_2.

Follow-up studies were done two to six months later on 94, or 50 percent, of this group of patients, and not one had evidence of any visual damage from methyl alcohol.

The blood CO_2 was 25 milliequivalents per liter or below in the remaining 132 patients. Thirty-seven of these patients died. All fatal cases had a severe acidosis with the blood CO_2 usually below 10 m.e.q./liter. Death was always characterized by cessation of respiration from medullary paralysis. Convulsions were common. The heart usually continued to beat for several minutes after respirations stopped.

When it was possible to obtain a history on the patients who died, it was learned that they were always blind or nearly blind before their final loss of consciousness. Ninety-

five patients with a low blood CO_2 survived the poisoning. Sixty-two of these patients had visual symptoms.

Objective ophthalmoscopic findings were present only in patients with visual symptoms and occurred in 87 percent of those. The relation of blood CO_2 before treatment to the final visual acuity showed that chances of restoration of complete vision was possible at almost any CO_2 level, but patients with a very low CO_2 frequently had permanent loss of vision. No patient with a CO_2 above 12 m.e.q./liter had a final visual acuity worse than 20/200 in either eye. These findings are illustrated graphically in Figure 4.

Treatment

Treatment was directed toward the elimination of acidosis in those patients with a low blood CO_2. Bicarbonate of soda was taken by mouth in patients whose symptoms were mild and whose blood CO_2 was between 20 and 25 m.e.q./liter. Patients with a very low CO_2 and severe symptoms were given soda bicarbonate intravenously.

The available supply of already prepared alkaline solutions for intravenous administration was quickly exhausted. New solutions were readily prepared by dissolving the dry powder from clean, previously unopened boxes of bicarbonate in flasks of sterile glucose, saline or distilled water.

Several hundred such solutions containing 25 to 50 gm. of bicarbonate per liter were given intravenously with no ill effects. Symptoms of alkalosis rarely developed even though the blood CO_2 rose to 40 or 50 m.e.q./liter in some patients.

Because of the strong tendency for acidosis to recur, it was found that alkali therapy had to be continued for a minimum of three days, with constant check of the blood CO_2 or urine pH. This was strikingly demonstrated in four patients who were treated in the emergency clinic and failed to follow instructions to take soda bicarbonate orally at home. They were brought back the next day in severe acidosis and died.

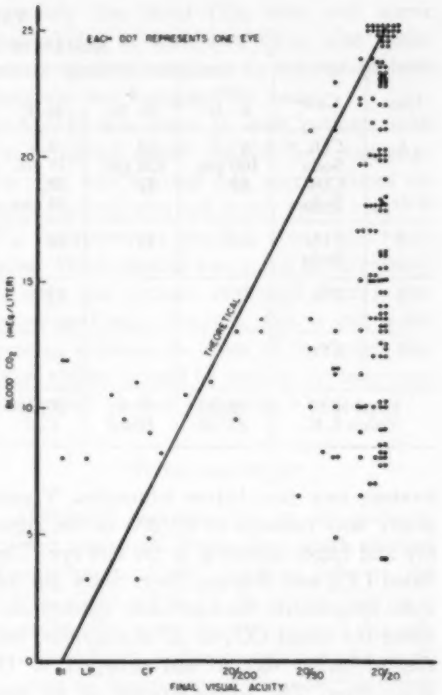

Fig. 4 (Benton and Calhoun). Correlation of initial blood CO_2 with final visual acuity. The wide scattering of the dots on the graph does not conform to the theoretic correlation represented by the diagonal line.

Adequate correction of acidosis in patients with severe methyl alcohol poisoning will not invariably save life or vision. If the acidosis is severe or has persisted for several hours, death may result in spite of energetic treatment, or, if the patient survives, he may have marked loss of vision.

Also, inadequate alkali therapy may result in a prolonged state of mild or moderate acidosis and permanent loss of vision. These observations are illustrated in Table 1.

Case reports

Cases 6, 7, and 8 demonstrated prolonged acidosis as the result of inadequate alkalinization.

Case 6. This Negro, aged 19 years, had consumed one-half pint of the methanol

TABLE 1

RESULTS OF ALKALI THERAPY

Day	Case	1 L. R.	2 D. N.	3 R. F.	4 L. S.	5 W. G.	6 R. H.	7 W. M.	8 W. R. M.
1	CO_2 Soda CO_2 Soda	4 100 gm. 42	8 120 gm. 52	9 35 gm. 30 20 gm.	3 40 gm. 33 30 gm.	5 75 gm. 26	9 60 gm. 27	13 50 gm. 30.5	15 50 gm. 22
2	CO_2 Soda	43	49	36	42	38	17 16 gm.	16 35 gm.	12 45 gm.
3	CO_2			23	45	16 30 gm.	28	22 20 gm.	19 30 gm.
4	CO_2					35			19 30 gm.
Final R.E. Vision L.E.		20/20 20/20	L. P. Blind	20/200 C. F.	C. F. L. P.	20/30 C. F.	20/20 C. F.	20/200 20/100	Blind Blind

mixture two days before admission. Visual acuity was reduced to 20/200 in the right eye and finger counting in the left eye. The blood CO_2 was 9 m.e.q./liter. Sixty gm. of soda bicarbonate intravenously quickly elevated the blood CO_2 to 27 m.e.q./liter, but the following day it had dropped to 17 m.e.q./liter. The administration of 16 gm. of soda by mouth returned the blood CO_2 to 28 m.e.q./liter. The final visual acuity was: R.E., 20/30; L.E., counting fingers at 18 inches. A centrocecal scotoma persisted in the left central field, whereas a pericecal scotoma in the right field spared the fixation area.

Case 7. This 26-year-old Negro drank one-half pint of the poisoned whiskey. Within 18 hours the vision became blurred. He did not seek treatment for another 18 hours, at which time his vision was reduced to finger counting at 10 inches. The blood CO_2 was 13 m.e.q./liter. Fifty gm. of soda bicarbonate intravenously elevated the blood CO_2 to 30.5 m.e.q./liter. The following day the blood CO_2 had fallen to 16 m.e.q./liter. Thirty-five gm. of soda by mouth brought the blood CO_2 up to 22 m.e.q./liter. The final visual acuity was: R.E., 20/200; L.E., 20/100.

Case 8. This 40-year-old Negro had consumed one-half pint of the methyl alcohol mixture. Dimness of vision was experienced after 12 hours. The blood CO_2 was 15 m.e.q./liter. For four days the blood CO_2 remained below 20 m.e.q./liter in spite of an initial dose of 50 gm. of bicarbonate of soda intravenously followed by oral doses of 30 to 45 gm. daily. Although this patient could count fingers accurately at 18 inches at the time of admission, he became and remained totally blind.

Cases 3 and 5 show the tendency for acidosis to recur after initial correction.

Case 3. This Negress, aged 34 years, became blind 48 hours after consuming one-half pint of the contaminated bootleg whiskey. The initial blood CO_2 was 9 m.e.q./liter. Soda bicarbonate was given intravenously in doses of 35 gm. later 20 gm. The blood CO_2 remained above 30 m.e.q./liter the following 36 hours and then fell to 23 m.e.q./liter. No further treatment was given.

The final visual acuity was: R.E., 20/200; L.E., counting fingers at six inches. A centrocecal scotoma persisted in the left field whereas the right fixation area was spared by a pericecal scotoma.

Case 5. This 37-year-old Negro drank "a little bit" of the methyl alcohol whiskey eight hours before being brought to the hospital. Amblyopia was severe and the blood CO_2 was 5 m.e.q./liter.

After 75 gm. of soda intravenously, the blood CO_2 became normal and remained so during the following day. Twenty-four hours later the blood CO_2 had dropped to 16 m.e.q./liter but was again elevated to normal by the oral administration of 30 gm. of soda. The final visual acuity was R.E., 20/40; L.E., counting fingers at 18 inches.

Again the difference in visual acuity between the two eyes was due to a centrocecal scotoma in the left eye and a pericentral scotoma in the right eye.

The poor vision in Cases 2 and 4 is evidence that adequate alkali therapy will not always prevent blindness.

Case 2. This 43-year-old Negro drank a moderate quantity of the poisoned whiskey over a two-day period. Blurred vision was experienced one day later. The patient was not brought to the hospital until the following day at which time he was blind. The blood CO_2 was 8 m.e.q./liter; 120 gm. of soda intravenously elevated the blood CO_2 to 50 m.e.q./liter where it remained for at least two days. In spite of this he gained only a little vision and then again became blind.

Case 4. A 47-year-old Negress drank one-half glass of the poisoned whiskey. There was headache, nausea, and vomiting in six hours followed the next day by blurring of vision.

The blood CO_2 at the time of hospital admission was 3.2 m.e.q./liter. After 40 and 30 gm. of soda intravenously the blood CO_2 remained over 40 m.e.q./liter for two days. The visual acuity improved in two months to 20/100, right eye, and finger counting at six inches, left eye, then dropped to finger counting in the right eye and light perception in the left.

Normal vision may result in the face of initially low blood CO_2 if treatment is prompt.

Case 1. This 20-year-old Negress experienced abdominal pain and poor vision 24 hours after drinking one-half glass of the bootleg whiskey. The blood CO_2 was 4 m.e.q./liter. After 100 gm. of soda intravenously the blood CO_2 was well above normal for at least two days. The visual acuity quickly returned to normal in both eyes and has remained the same.

ACTH was given to four patients with no beneficial effect. Spinal fluid exchange therapy was carried out several times on three patients who had severe visual loss.[10, 16] No improvement resulted from this treatment. Ethyl alcohol was given intravenously to only one patient. Although definite improvement was observed, this is not given here as evidence in favor of the belief that ethyl alcohol should be used in the treatment of methyl alcohol poisoning.[1, 2, 14]

Histologic study

Histologic examinations on six eyes removed six to 18 hours after death showed degeneration of the ganglion cells of the retina, slight edema of the nerve-fiber layer near the disc, some degeneration of the rod-and-cone layer, with accumulation of a small amount of albuminous fluid under the retina near the disc.

All these changes could have been the results of normal post-mortem degeneration, particularly since the changes were more prominent in those eyes removed at 18 hours than in those removed at six hours after death. Other investigators have presented considerable evidence that degeneration of the retinal ganglion cells is a specific toxic effect of methyl alcohol.[6, 8, 14]

Other pathologic findings of interest were a mild to moderate edema of the brain and an acute pancreatitis. Studies of cerebral blood flow were done on five patients with severe poisoning. The average arterial oxygen saturation was 92 percent. Cerebral blood flow was reduced 33 percent and cerebral oxygen consumption was diminished 35 percent. Serum-amylase determinations on several patients showed moderate to marked high levels. It was felt that acute pancreatitis was often the cause of the severe abdominal pains in these patients.[5]

Toxic action

Many theories have been advanced to explain the mode of toxic action of methyl alcohol in the human body.[6, 9, 13-15] It is unfortunate that laboratory animals do not respond to this agent in a manner similar to the human. Acidosis almost never develops and the animals do not often appear to go blind. Fink[6] and also McGregor[9] have described degeneration of the retinal ganglion cells in animals poisoned with methyl alcohol, but this has been denied by Roe.[14] Conclusions from experiments on animals should be applied to humans with great caution and reservation.

Apparently methyl alcohol breaks down slowly in the body to formaldehyde and formic acid. It appears unlikely that any of these chemicals acts directly on the retina or other nerve tissues. Methyl alcohol is rapidly distributed throughout all body fluids but symptoms of poisoning rarely develop in less than 18 hours after ingestion.

According to the theory of Rohr,[13] methyl alcohol or one of its breakdown products slowly combines with the iron catalyst of the oxydase system rendering it inactive and causing a tissue anoxia. This process is accelerated in the presence of acidosis.[16]

Because the retina has the highest oxygen requirement per unit concentration of iron of any body tissue, it is not surprising that this delicate ocular membrane should be damaged in methyl-alcohol poisoning. An indirect toxic action, as just described, offers a ready explanation for the parallelism of ocular symptoms and signs of methanol poisoning with the amblyopia of lead, arsenic, thallium, or carbon disulfide poisoning or even that of severe malnutrition.

Recently, Potts and Johnson[9] found evidence that the proximal toxic agent in methanol poisoning may be formaldehyde, a potent inhibitor of retinal oxidation and glycolysis, and expressed the belief that the role of acidosis was a highly questionable one.

In methyl-alcohol poisoning, the acidosis does not appear to be the cause of amblyopia but it does act as an accelerating force. If acidosis can be corrected before permanent ocular damage has resulted, a return of normal visual acuity can be expected. If retinal hypoxia has existed long enough for irreversible degeneration of the ganglion cells to occur, treatment may prevent death but visual impairment will be permanent.

Until a specific chemical is found that will prevent or release the combination of methyl alcohol or its products with the retinal enzyme systems, rapid, intense, and prolonged alkalinization appears to be the most important phase of treatment for methyl alcohol poisoning.

Summary

Clinical and pathologic observations on a group of 320 patients with acute methyl alcohol poisoning are reported. There were 37 deaths.

Visual symptoms, occurring 18 to 48 hours after ingestion of methanol, included blurred vision, loss of central vision, and complete blindness. The pupils reacted sluggishly or not at all to light. Absence of the pupillary light reflex was a sign of poor prognosis for life or eventual restoration of visual acuity.

The visible retinal changes were hypermia of the optic nervehead and superficial edema of the papilla and surrounding retina, and later a simple atrophy of the optic nerve. All patients with severe retinal edema had some degree of permanent visual loss.

The earliest central field defect was usually a centrocecal scotoma. Later more complex types of visual defects appeared.

Acidosis was present in every severely poisoned patient. The degree of acidosis paralleled the severity of general and ocular symptoms.

Partial or complete recovery of the initially reduced visual acuity was observed in most patients. If the visual acuity did not

return to normal within six days after treatment was begun, it invariably dropped again to a very low level. The final vision was 20/200 or less in both eyes of six patients and in one eye of three patients.

Histologic studies on six eyes enucleated after death revealed only changes consistent with post-mortem degeneration.

On the basis of results obtained in this series of cases, the most effective treatment of acute methyl alcohol poisoning consists of intensive and prolonged alkalinization.

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THERAPEUTIC VALUE OF NEBACETIN IN OPHTHALMOLOGY*

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An attempt has been made to estimate the efficacy of nebacetin† for ophthalmic purposes. Only topical application of nebacetin has been attempted. Experiments to determine the tolerance of the posterior and central tissues to nebacetin are, as yet, not completed.

Present investigation

The ophthalmic value of nebacetin was investigated in two ways:

I. Normal bacterial flora

We examined the effect of nebacetin on the bacterial flora occurring normally in the conjunctiva. Total asepsis of the conjunctiva cannot, of course, be obtained by any means.

Thirteen patients were given penicillin drops in the right eye and nebacetin in the left. The instillations were begun about noon and were given at intervals of two hours, that is, five or six times that day; two additional instillations were given the next morning. Cultures were taken from the conjunctiva one to two hours after the treatment and were sent to the Department of Bacterial Resistance, State Serological Institute, for bacteriologic examination.

Results

The results are reported in Table 1 which gives the character and number of bacterial strains obtained in the cultures before and after the antibiotic treatment of the conjunctival space. As will be noticed from these results, the use of nebacetin proved highly successful, as it reduced the bacterial content of the conjunctiva even after short treatment.

Prior to treatment, the cultures showed a bacterial content of 25 strains; after treatment, only four staphylococcal strains were present. Out of 13 cultures made after treatment, nine showed no growth whatever.

The corresponding figures for the penicillin treatment are 27 strains before and 13 after treatment, including three out of 13 cultures without any growth. As was to be expected, the gram-negative strains, which are not sensitive to penicillin, disappeared under treatment with nebacetin.

According to these experiments, nebacetin should prove particularly effective as a preoperative agent. For one thing, in order to combat fresh bacterial invasion, the drops may be used until immediately before an operation, since this preparation does not give rise to any conjunctival irritation or congestion. Furthermore, according to other authors, as well as our own experiences, nebacetin seldom gives rise to allergic reactions.

II. Treatment of external eye diseases

Before applying nebacetin to patients, we used the solution, as well as the ointment, on ourselves and on a few volunteers. If the eye drops are too cold, they cause a little conjunctival smarting. However, if they are at room temperature they give no inconven-

TABLE 1

Number of bacterial strains present in a normal conjunctiva before and after treatment with penicillin and nebacetin

	Penicillin Treatment		Nebacetin Treatment	
	Before	After	Before	After
Staph. aureus	5	1	4	0
Staph. albus	12	7	12	4
Strep. nonhem.	5	0	6	0
Corynebact.	3	1	0	0
Klebsiella	1	1	2	0
Enterococci	1	1	1	0
Total	27	11	25	4

* From the eye department of the Royal Danish Military Hospital.

† Nebacetin for instillation: 5.0 mg. neomycin plus 250 units of bacitracin per cc. Nebacetin ointment: 5.0 mg. neomycin plus 250 units bacitracin per gm.

TABLE 2

THERAPEUTIC RESULTS WITH NEBACETIN

	Cured or Decidedly Improved	No Effect	Worse	Total
Acute external eye diseases				
Acute conjunctivitis	32	5	0	37
Acute keratoconjunctivitis	22	3	1	26
Dacryocystitis	8	0	0	8
Palpebral abscess	2	0	0	2
	64 (80%)	8 (11%)	1 (1, 7%)	73
	Improved	No effect	Worse	Total
Chronic external eye diseases				
Chronic conjunctivitis	15	4	2	21
Blepharoconjunctivitis	26	1	3	30
	41 (80%)	5 (10%)	5 (10%)	51

ience. The ointment causes no inconvenience at all, being of a consistency that makes it particularly useful for ophthalmic treatment.

Not until we had commenced the experiments here reported was collaboration established between our clinics and the Department of Bacterial Resistance of The State Serological Institute. Consequently, bacteriologic examinations and determinations of bacterial resistance have been available for only 67 percent of the joint material.

In ophthalmic practice, it is a common rule to employ antibiotics in such a form for out-patient treatment that the prescription covers a period of four days. After this the patient returns for control examination, and the prescription is altered according to the therapeutic effect obtained.

Thus, for instance, instillations every hour are prescribed for four days, and the next time the instillations may be reduced to four times a day for the following four days and so on. We have chosen to employ nebacetin in this way.

In a considerable number of cases, failure to return for control examination, irregular use, and discontinuation of treatment for some nonclinical reasons (bottle broken) have resulted in these cases being excluded from the material.

The present material, therefore, comprises a total of 124 patients with external eye diseases who have been treated with nebacetin under adequate control, and whose therapeutic results can be estimated with a high degree of certainty. In addition, our material also includes a few cases unsuitable for the classification here adopted. These cases will be mentioned separately.

The therapeutic results in cases of acute external eye disease have been classified as: (1) Cured or decidedly improved; (2) no effect; (3) worse.

For the chronic cases of external eye diseases the following classification has been chosen: (1) Improved; (2) no effect; (3) worse.

In this group of patients, the term "improved" means mitigation of the complaint and/or objective improvement in the form of diminution or disappearance of congestion or secretion—that is, the patient has been relieved of the symptoms which made him seek ophthalmic advice. In reality, we do not think we have obtained any recovery from chronic external eye lesions by treatment with nebacetin.

Table 2 gives the results of therapy with nebacetin.

Various groups of patients treated at the Ophthalmological Department of the Royal

Military Hospital and the private clinics of the authors showed a striking percentage uniformity, even though they were collected by different ophthalmologists.

In one case of phlyctenular keratitis treated with nebacetin, the ulcer became clean but failed to heal. Recovery was later obtained with cortisone. A case of herpetic keratitis was likewise cleaned by application of nebacetin, but it required other treatment before it healed. A case of dendritic keratitis was getting worse under treatment with nebacetin. In this case the lesions proved to be due to mycotic infection.

The bacteriologic reports received from the State Serological Institute showed no clear connection between the bacteriologic findings and the clinical results. Among 11 cases of acute conjunctivitis in which cultures were made before, during, and after the treatment, 10 patients with the cultural result of "no growth" recovered, and one recovered in whom the cultures showed scanty growth of staphylococci.

Among five cases of acute keratitis, three with a cultural result of "no growth" recovered, and so did two patients with a cultural result of staphylococci.

Among eight cases of dacryocystitis the lacrimal sacs of five were rinsed with nebacetin. No pneumococci were demonstrated in the lavage (the first lavage was performed with sterile water, part of which was sent to the Pneumococcus Department of the State Serological Institute). Two patients showing a cultural result of no growth recovered, while three patients with growth of various bacterial strains also recovered.

The present material comprises a total of 170 resistance determinations. None of the cases showed any evidence of any bacterial type becoming resistant to bacitracin or neomycin under this treatment.

Four patients gave an immediate allergic reaction to nebacetin. One had acute conjunctivitis. The remaining three belonged to the group of chronic external eye diseases, with case histories in which positive allergic reaction to atropine, zinc, mercury, noviform, sulfonamides, and so forth, had been registered.

In two cases, recorded as "worse," the nebacetin therapy had to be discontinued because these patients complained of intense burning unaccompanied by any objective symptoms.

Summary

The therapeutic value of nebacetin in ophthalmology has been investigated in two ways:

1. In 13 cases one eye was treated with penicillin, the other eye with nebacetin every second hour for a day. Bacteriologic examinations were made before and after treatment.

Penicillin treatment reduced the number of bacterial strains from 27 to 11.

Nebacetin reduced 25 strains to four.

Asepsis resulted from penicillin treatment in three cases, from nebacetin treatment in nine cases.

2. Seventy-three cases of acute external diseases were treated with nebacetin (acute conjunctivitis, keratitis, dacryocystitis). Sixty-four (88 percent) were cured; in eight cases (11 percent) no effect was observed; a single case (one percent) deteriorated.

Fifty-one cases of chronic external diseases (chronic conjunctivitis and blepharoconjunctivitis) responded to nebacetin treatment as follows: 41 (80 percent) were relieved or objectively improved. No effect was seen in five cases (10 percent), five cases (10 percent) deteriorated.

Bacteriologic examinations and resistance tests were made at the Danish State Serological Institute. No conformity between bacteriologic and clinical finding was established.

THE CLASSIFICATION AND TREATMENT OF ALLERGIES OF THE CONJUNCTIVA*

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Ever since the importance of allergy as a disease mechanism gained general acceptance and the many and varied manifestations of hypersensitivity became fully recognized, ophthalmologists have been quick to appreciate the major role that allergy plays in diseases of the eye. This has proved particularly true in regard to allergies of the outer eye.

Yet, even today, because allergic conjunctivitis is not a single entity but, instead, assumes many forms, difficulties in diagnosis still arise. Moreover, since certain types of allergic conjunctivitis may resemble conjunctivitis of nonallergic origin, such as bacterial and viral infections, on many occasions the diagnosis of allergic conjunctivitis is either not entertained when allergy occurs, or else erroneously made when no allergy exists. As a result, serious problems in the management of these patients may arise.

If, however, the conjunctival manifestations of allergy are correlated and classified in accordance with the allergic mechanisms involved, many otherwise troublesome diagnostic and therapeutic problems are resolved. It is therefore the purpose of this paper to attempt such an organized exposition of the subject of conjunctival allergy based both on general allergic concepts and on clinical and experimental experiences.

GENERAL ALLERGIC CONCEPTS

IMMUNOLOGIC ASPECTS OF ALLERGY

Allergy is considered a specifically altered capacity to react as judged from previous experience of the same individual or from experiences of other individuals of the same species. The term is generally used to denote acquired hypersensitivity, although tendencies to develop allergy are influenced by heredity. Atopy is the term used to designate allergy with a hereditary background; that is, a familial history of allergy.

The concept of hypersensitivity, on an immunologic basis, is less than 50 years old, having first been introduced as "anaphylaxis" by Richet,[1] in 1902. It was confirmed independently by Theobald Smith,[2] who noted the reaction with horse serum just about the same time. The name allergy was coined by Van Pirquet,[3] in 1906, and has been used ever since because it is so neat an expression.

For lack of a name or a theory, the faultless experiments of a clinician, Blackley,[4] in 1873, demonstrating that pollen caused hay fever, had been disregarded. Thus, it came about that bacteriologists assumed control of research in this new field and have influenced its development along certain lines, emphasizing a fundamental antigen-antibody philosophy. Only recently has this approach been challenged.

ALLERGENS

Substances that can cause allergic states and, thereafter, allergic reactions, are almost limitless. Besides foreign proteins, foods and digestive products, and nitrogenous materials as in pollens, the list includes chemicals, drugs, metals, living or dead micro-organisms (molds, yeast, fungi, bacteria, viruses, and some of their soluble constituents and metabolites). Physical influences such as light, heat, and cold, as well as emotional factors, also cause allergic responses.

It is thus obvious that while some of these substances are true antigens, and others, such as pollens, show some degree of antigenicity, many are frankly not antigens in the usual sense. Many of these allergic ex-

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citants or allergens are believed to combine with proteins or other substance of the host's tissues and to assume thereby the nature of an artificial antigen, thus causing allergic reactions.

Allergic responses

It is convenient to divide allergic reactions into two types: (1) The immediate, and (2) the delayed. While the distinctions are not always clear cut and some overlapping occurs, this classification is of great value.

I. *Immediate reactions*

"Immediate" allergic responses occur shortly after application or absorption of the allergen. It is usually a matter of minutes, at most an hour. Immediate reactions occur in asthma, hay fever, hives, unusual cases of drug sensitivity, and gastrointestinal disturbances, and are usually due to serum and other proteins, danders, pollens, dusts, or foods. The reaction is characterized by:

1. Skin tests give a typical wheal in a few minutes.
2. A drop of the allergen in the eye is followed promptly by itching, tearing, redness, and chemosis.
3. Circulating antibodies, such as precipitins, are often demonstrable.
4. Passive transfer (Prausnitz-Kuestner phenomenon) occurs.
5. Desensitization following the injection of minute quantities of antigen is often successful, and results in the formation of "blocking antibody" which combines readily and specifically with the allergenic material and "neutralizes" it.
6. Histamine plays a dominant role in the production of the reaction.

The Prausnitz-Kuestner reaction is so important in allergy as to deserve a little more elaboration. Kuestner was very sensitive to fish. He injected a little of his serum into the skin of Prausnitz. Twenty-four hours later an extract of fish injected into the site, so prepared, caused a marked local reaction (wheal) which soon faded. Extension of this original work has revealed that the reaction may occur at a sensitized site, not only when the allergen is introduced locally, but also when it is injected in remote tissues, or into the blood stream, or is absorbed following ingestion or inhalation.

These antibodies are known as "reagins" or "skin-sensitizing" antibodies. In addition to the skin, the mucous membranes of the eye, nose, and intestinal tract are capable of similar passive sensitization.

II. *The delayed type of response*

The delayed type of allergic response is also known as the "tuberculin or bacterial type" because reactions do not become manifest for at least several hours, and usually not until 24 to 48 hours after antigen contact. It generally occurs in drug or chemical allergy, especially contact dermatitis, and in bacterial allergy, that is, allergy to all types of micro-organisms and their products.

In contradistinction to the immediate type of allergic response, the delayed type is characterized by:

1. Absence of circulating humoral antibodies. The cells themselves are the seat of the antigen-specific reaction.
2. Inability to produce passive transfer of the allergy.

While most drug allergies and other types of contact dermatitis give the delayed type of reaction, anaphylactic and immediate reactions may sometimes occur, as well as passive transfer of serum, especially if the sensitization is sufficiently intense.

In bacterial allergy, in addition to the usual delayed type, at times an immediate type of allergic reaction does occur. In some cases it is due to the nucleoprotein, in other instances to the polysaccharide fraction.

Present-day thought on the relationship of bacterial allergy to immunity tends to favor the idea that they are not parts of the same process of protection. They may exist together or separately. Immunity is a protective process, while allergy is an inflammatory and necrotic process which is essentially

harmful. As Rich[5] has put it, "allergy converts a bacteria which does not produce a toxin into one that does, i.e. it converts a harmless protein product of bacterial disintegration into a violent and lethal poison."

Recent ideas on allergy

In the brief review just given of the immunologic aspects of allergy, emphasis has necessarily been placed on antigen-antibody mechanisms. This approach, however, does not explain many phenomena in the field of allergy and even the immunologically trained allergist is often obliged to make diagnoses solely on a clinical basis. For this reason, recent thought has utilized the contributions of Selye[6] concerning the general adaptation syndrome, which is based on pituitary-adrenal physiology, in order to explain less well understood allergic reactions. Additional impetus to this line of thought has been given by the introduction of ACTH and cortisone and the beneficial results that have been noted in their use in various allergies and related conditions.

Williams,[7] basing much of his thought on Selye's work, has defined allergy as an inherited predisposition to a localized type of autonomic dysfunction mediated by cholinergic fibers of the autonomic system. Thus, a minor stimulus may cause a reaction resulting in cellular damage, the release of histamine, and vascular reactions, causing a typical allergic picture. An antigen-antibody reaction may be associated with the vascular reaction, but it is a secondary phenomenon, phylogenetically more recent than the vascular component of the autonomic reaction.

Allergic reactions of the conjunctiva

It has been shown experimentally that not only may the conjunctiva be sensitized like other tissues, participating in the general hypersensitivity following parenteral injection of antigen, but that it has the propensity of reacting very markedly to local contact with the allergen. This reaction has provided the allergist with a rather dramatic, if sometimes hazardous, method of testing sensitivity, particularly for horse serum.

As Woods[8] has so clearly stated it, the eye will develop local allergic inflammatory reactions if, (1) in the generally sensitized body, the antigen is later brought in direct contact with the ocular tissues, or, (2) if an increased local hypersensitivity has already been produced by primary sensitization of the eye and the antigen later reaches the eye from systemic absorption.

Unless the conjunctiva has been specially sensitized by direct contact, later systemic absorption of the allergen usually produces no conjunctival reaction. Thus, in most cases, all allergic conjunctival reactions imply that at one time or other, the conjunctiva has been exposed to direct contact, by inoculation or infection, with the allergen.

An interesting experiment reported by Chait[9] deserves mention. In a child with complete dacryostenosis, an intradermal injection of serum from an individual who was very sensitive to peanuts, was made. After 24 hours a pledget of cotton soaked with peanut extract was placed in the child's lower conjunctival sac. A reaction appeared at the sensitized area of skin only 21 minutes later. This indicates that eye drops may cause an allergic systemic reaction.

Direct contact of allergens with the conjunctiva is a common occurrence. The conjunctiva is readily accessible to air-borne allergens, has an exposed position to contactants, is frequently the site of administration of drugs, and is repeatedly infected by bacteria. It is no surprise, therefore, that allergic conjunctivitis is frequently encountered.

Clinical manifestations of allergic conjunctivitis

The recognition and management of the various types of conjunctival allergy that are encountered in practice is greatly facilitated by a classification such as that offered in Table 1. Excluding vernal conjunctivitis, which is not discussed in this paper as it

TABLE 1

CLASSIFICATION OF CONJUNCTIVAL ALLERGIES

1. ACUTE ALLERGIC CONJUNCTIVITIS
   Atopic (familial history of allergy)
   Immediate allergic response (in a few minutes)
   Positive intradermal test in 15 to 30 minutes
   Due to inhalants, pollens, dusts, danders, feathers, or foods
   Major characteristic: chemosis

2. ALLERGIC DERMATOCONJUNCTIVITIS
   Delayed allergic response (24 to 48 hours)
   Positive patch test in one to five days
   Contact allergy due to drugs, cosmetics, chemicals, or apparel
   Major characteristic: eczema

3. CHRONIC ALLERGIC CONJUNCTIVITIS
   A. *Atopic type*
      Immediate allergic response
      Positive intradermal test in 15 to 30 minutes
      Due to inhalants, pollens, danders, dusts, feathers, or foods

   B. *Bacterial type*
      Delayed allergic response
      Delayed positive intradermal test (24 to 48 hours)
      Due to bacteria, fungi, and possibly viruses

belongs in a separate category, this classification, as will be developed below, would appear to cover all other manifestations of allergic conjunctivitis.

ACUTE ALLERGIC CONJUNCTIVITIS

This type of hypersensitivity is characterized by a sudden or immediate hyperemia and edema. Marked chemosis of a "glassy" appearance,[10] associated usually with a nonpurulent watery discharge, occurs. It is the conjunctival prototype of the immediate type of allergic response already described and usually is caused by the air-borne group of allergens, pollens, animal danders and feathers, mold spores, and dusts. The conjunctival reaction is similar to the general inflammatory edema which involves the upper respiratory tract.

A similar picture may occur in food allergies, such as egg and milk, but in that event the ocular reaction has only a minor role in a dramatic general systemic reaction. A familial history of allergy (atopy) is generally obtained.

As in the nasal symptoms of hay fever, the reaction may be so intense that a profuse mucopurulent discharge occurs. Itching and burning may be intolerable. Eosinophils often occur in abundance in the secretions. Vasoconstrictors, like epinephrine, neosynephrin, and privine help control the reaction which usually subsides rapidly once the excitant is removed.

Skin tests of the intradermal type give an immediate wheal response in most cases of this group. Ophthalmic tests, by means of the direct instillation of the suspected allergen in weak dilution, are generally not safe in these cases. Severe, and possibly permanent, ocular damage may result. Because of chemosis early epidemic keratoconjunctivitis may be confused with acute allergic conjunctivitis. The virus infection, however, is differentiated by the presence of preauricular lymphadenopathy. On scrapings, mononuclear cells are found instead of eosinophils.

ALLERGIC DERMATOCONJUNCTIVITIS

The eczematous exudative type of allergic conjunctivitis is perhaps the most common type encountered in ophthalmic practice. It is essentially a form of contact allergy, usually due to the use of drugs, cosmetics, articles of clothing and jewelry, or chemicals used in industry. Unless the individual has been previously sensitized, the allergy generally occurs only after prolonged use of the offend-

ing substance. The allergic response almost always is of the delayed type.

The clinical picture consists of a papillary conjunctivitis which may be very severe. Some chemosis may occur. While the discharge often is watery, in many cases it may be sufficiently mucopurulent to resemble bacterial conjunctivitis. Eosinophils and basophils are found in many, but not all, cases, especially if the reaction has been present for several weeks or more.

The outstanding feature, present in all but the mildest cases, is the involvement of the skin of the eyelids. This is characterized by dryness, thickening, abnormal wrinkling, or frank eczema, sometimes, as with the local use of penicillin, being very marked. Either the skin of the upper lid or of the lower lid may alone be involved, as well as both together. This often provides a clue as to the etiology.

When an allergic conjunctivitis is due to a drug, especially eye-drops instilled in the lower cul-de-sac, the earliest skin involvement may follow the usual paths of tear overflow, that is, the canthi and the adjacent lower lid skin. Ointments often produce changes along the lid margins, which are swollen and inflamed, but do not exhibit the scales and ulcers seen in typical blepharitis. Allergy due to cosmetics, especially nail polish and hair tonics or rinses, on the other hand, often first involves the skin of the upper lids.

Unlike the sudden edematous type of allergic conjunctivitis, here the conjunctival reaction usually takes hours to several days to occur, even after sensitization has taken place, and disappears relatively slowly, in a matter of days or a week, after the offending substance is eliminated.

The diagnosis in allergic conjunctivitis due to drugs is often easy. The clinical appearance, the history and the finding of eosinophils in the secretion, followed by the beneficial effect of the elimination of the causal agent, makes a satisfying sequence of events. The identification of other cutaneous and mucosal sensitizers usually requires more effort.

It is important to differentiate allergic dermatoconjunctivitis from infectious eczematoid staphylococcal dermatitis. This latter condition may give a clinical picture resembling allergy very closely as the eczema, while presumably due to the dermo-necrotizing toxin of the staphylococcus, may represent an allergic response to this toxin. Many cases of this type of staphylococcal infection are treated for long periods of time as cases of allergy. The intensive antistaphylococcal regime, including toxoid injections, required to cure the condition is entirely different from that used in contact allergy.

Infectious eczematoid staphylococcal dermatitis as differentiated from allergic dermatoconjunctivitis shows: (1) Blepharitis, often ulcerative; (2) a drier type conjunctivitis; (3) superficial erosions of the corneal epithelium on slitlamp examination; (4) numerous toxic staphylococci when cultures are taken; (5) absence of eosinophils in epithelial scrapings.

Primary irritants versus *Cutaneous sensitizers*

It was explained above that the group of nonprotein allergens under discussion are not primarily antigens, but, probably by union with the host tissues, form antigenic compounds. Substances that come in contact with the skin and mucosa may be classified as (1) primary irritants and (2) cutaneous and mucosal sensitizers.

Primary irritants cause an inflammatory reaction by direct action on the site of contact only, if allowed to act in sufficient intensity or quantity for a long enough time. The eyelids, being so thin, are more susceptible to such irritants than parts of the body where the skin is thicker. The frequency of burns due to the use of even weak solutions of iodine in the preparation of patients for eye surgery is a case in point.

Persons with dry skins are especially susceptible to dehydrating chemicals. The ma-

jority of occupational dermatoses are not allergic, but occur in workers with anatomic or physiologic skin defects, or upon excessive contact in those who do not take proper precautions.[11]

Primary irritants may also be sensitizers. Cutaneous and mucosal sensitizers do not necessarily cause changes on first contact, but after five to seven days or more the entire skin, or all the mucous membranes, will react specifically on further contact at the original site or elsewhere. These are the sensitizers that cause what is known as allergic contact dermatitis or contact allergy.

For many years it has been believed that contact dermatitis represented rare individual and innate idiosyncrasies. That this idea is untenable was shown by Bloch and Steiner-Wourlisch,[12] in 1926, when they proved that any individual could be made sensitive to the primrose plant, if one used a concentrated enough extract a sufficient number of times. Some persons needed only one exposure, others a number. In this regard, perhaps heredity played a role.

In order to prove contact allergy patch tests are employed. Immediate wheal testing by intradermal injection is not a correct diagnostic procedure. A patch test consists of applying a small amount of the suspected substance to a site of normal skin of the patient and covering it with an innocuous impermeable material which is sealed to the skin with adhesive and left in site for one to three or four days. Sometimes one must read the reactions for as long as five days after the patch is removed, as it may only then become positive. Usually 24 hours' contact is enough. The true allergic reaction increases (as a rule) in intensity for 24 to 48 hours after the patch is removed.

A negative patch test does not necessarily rule out the test substance as a causative agent, because (1) under the test circumstances, the actual mechanism producing the inflammation may be lacking, (2) the patient may no longer be sensitive, (3) the actual sensitizer is not applied, and (4) a local sensitivity, only, is present, and the site of skin tested is not sensitive.

It is important that the test material not be too concentrated, as then it may act as a primary skin irritant. For testing liquids, a piece of gauze one-fourth inch square is saturated. Powders are placed on moistened gauze. For solids insoluble in water, make a saturated solution in a solvent, wet some gauze with the solution, but let the gauze dry first before applying it to the skin to eliminate the action of the solvent. For ointments, the same procedure as for liquids is used.

Ophthalmic tests, especially for drug allergies, should be done rarely and then with great caution, using very weak concentrations, as deleterious effects may occur.

Important ocular contact allergens

As far as the eye is concerned, the most important causes of contact allergy are ophthalmic drugs and cosmetics. Less commonly, articles of apparel, jewelry, and metals, such as nickel (used in spectacle frames), cobalt, platinum, chromium, and gold are responsible. Plastic spectacle frames may also cause allergies.

Most ophthalmic drugs are true sensitizers. Contact allergy is most frequently encountered in the following groups:

Local anesthetics

Apparently every local anesthetic, used for instillation, is a potential sensitizer and has been known to cause allergic conjunctivitis. Of the commonly used ones, those least likely to do so appear to be cocaine, phenacaine (holocaine), and, possibly, metycaine. Pontocaine, butyn, nupercaine, and larocaine are particularly likely to cause reactions, and frequently are the cause of cases of conjunctivitis that get progressively worse despite treatment.

Antibiotics

Penicillin is notorious for its sensitizing tendencies, especially in the ointment form,

a fact that has almost brought it into disuse for topical administration. It may be that penicillin sensitivity, upon topical use, is so common because of previous sensitization of persons, especially adults, to related fungus antigens such as trichophytin.[13]

Children are less sensitive to penicillin, as they are to trichophytin. Ocular reactions usually resembling angioneurotic edema have occurred following injection of penicillin intramuscularly. Reactions such as this, as well as urticaria following the use of penicillin, may be due to a mechanism similar to that of serum sickness.

Contact allergy to streptomycin, another frequent and potent sensitizer, usually occurs in nurses as a result of handling the drug.

Bacitracin appears to be the least sensitizing of all the other antibiotics, although allergies from it do occur. It may be that antibiotics derived from bacteria, as bacitracin is, are less allergenic than those derived from fungi. Thus sensitivities to aureomycin, chloramphenicol, and terramycin, when used locally, appear to occur more frequently. Neomycin and polymyxin appear, however, to be less allergenic.

Sulfonamides

All sulfonamides are prone to cause allergic reactions after prolonged use. The reactions are rarely as severe as those with the antibiotics, although sometimes they can be even worse. Sodium sulfacetimide and gantrisin cause fewer allergies than the original drugs in this group, such as sulfanilamide and sulfathiazole.

It should be emphasized that the indiscriminate use of such highly allergenic drugs as the antibiotics and sulfonamides not only results in unwarranted local sensitivities, but may also cause severe generalized allergies. Sometimes the use of that particular medicament, or related drugs, in any form, will be contraindicated thereafter.

Mydriatic and miotic alkaloids

Allergy to atropine is a common occurrence, resulting in a typical allergic dermatoconjunctivitis with eczema, papillary conjunctivitis, and conjunctival eosinophilia. Both hyoscine and homatropine may also cause sensitivities, but the eczematous component is less evident. Atropine, however, sometimes causes follicular conjunctivitis without eosinophilia or dermatitis due to conjunctival irritation (see later in article).

The miotic alkaloids, on the other hand, are rarely sensitizers. The use of pilocarpine or eserine only occasionally results in true allergic dermatoconjunctivitis. Instead, the prolonged administration of these and other related synthetic miotics may produce follicular conjunctivitis, without eczema and eosinophilia, which appears to be due to drug irritation,[14] not to allergy. This will be elaborated upon later. Those very rare instances in which allergic dermatoconjunctivitis as well as follicles occur, are explained by the fact that primary irritants may also be, or can become, sensitizers.

The syndrome of drug irritation

Although most cases of drug intolerance are due to drug allergy, enough instances of drug irritation occur to make it an important consideration. Before the concept of allergy was introduced drug intolerance was generally attributed to some form of irritation.

Nowadays because of the importance of allergy in our thinking, not enough emphasis is being placed on the fact that drug irritation still is a distinct entity. Actually in the case of certain medicaments, such as the miotic alkaloids, irritation to preparations of these drugs occurs much more frequently than true allergy.

While some drugs are themselves primary irritants, and almost every drug may at times be irritating, conjunctivitis due to drug irritation usually occurs following prolonged use of preparations that are prone to deteriorate into irritating degradation products if not prepared in a manner that will hinder or prevent this break-down.

The clinical picture of irritative conjunc-

tivitis due to drugs is that of a nonspecific watery conjunctivitis in which, in contrast to allergic dermatoconjunctivitis, there is neither dermatitis of the eyelids nor conjunctival eosinophilia. In chronic cases, especially with the use of the alkaloids, follicular conjunctivitis may be the outstanding feature. The conjunctival involvement is less diffuse and less uniform than in allergy; often the area of most irritation, that is the lower lid and adjacent bulbar conjunctiva, bears the brunt of the involvement. Skin-patch tests are negative with these low-grade irritants.

Mild chronic drug irritation must be differentiated from acute reactions due to strong chemical and vegetable irritants, such as lime, ipecac, insect powders, ricin, and turpentine oil, that may enter the conjunctival sac accidentally or because of malingering. Here severe reactions or burns occur, along with massive eosinophilia[15] and patch tests may be positive.

Drug irritation may generally be avoided or eliminated once it has manifested itself, by the use of preparations of the same drug that do not break down to the irritating end-products responsible for the irritation.[14] When, on the other hand, drug allergy occurs, the offending drug, and even related products, must be avoided.

Thus, it is important therapeutically to distinguish drug irritation from drug allergy.

Follicular conjunctivitis following use of the miotic and mydriatic alkaloids

Follicular conjunctivitis occurring with the use of atropine, pilocarpine, and eserine were considered by Morax[16] as instances of allergy and classified by him as instances of "toxic follicular conjunctivitis." Later Thygeson[17,18] pointed out that reactions from pilocarpine and eserine differed from true allergic reactions because of, first, the absence of dermatitis and eosinophilia, and, second, the presence of follicles which are so rare in allergic dermatoconjunctivitis. On this basis, he felt they should be considered as toxic reactions.

It would appear, however, that the follicular conjunctivitis that occurs with the use of preparations of the miotic and mydriatic alkaloids as well as related synthetic miotics is the result of chronic irritation due to the formation of irritating end-products by deterioration and degradation.[14] Alkaloids as a group are particularly prone to do this.

Thus, atropine, hyoscine, and homatropine form tropic acid, mandelic acid, and tropine. Pilocarpine forms acetic acid, propionic acid, ammonia, and methylamine. Eserine breaks down to rubreserine, eserine brown, eserine blue, and methylamine. Prostigmine, which is rather stable, degrades to dimethylamine. Di-isopropyl fluorophosphate forms hydrofluoric acid.

I have encountered follicular conjunctivitis without dermatitis and eosinophilia with the use of all these drugs. It therefore seems to me that the basis for the irritative follicular conjunctivitis characteristic of the ophthalmic alkaloids is the formation of irritant degradation products. It is interesting to note that all the miotic alkaloids and the synthetic miotics already mentioned, as well as Mecholyl and Carbachol, contain methyl ammonium radicals which apparently break down to methylamine or dimethylamine.

Other common ophthalmic drugs

Mercurials are well-recognized sensitizers, as are drugs ranging from dionin to boric acid, including zinc salts, argyrol, and privine. Even topical cortisone may cause an allergy.

Ophthalmic vehicles

Preservatives used in eye solutions may be the unsuspected cause of allergy. This especially applies to the mercurials. Other preservatives such as benzalkonium chloride, and possibly chlorobutanol, generally cause primary irritation rather than allergy.

Ointment bases are often not tolerated by patients, possibly due to sensitivity to lano-

lin or petrolatum. Here high-viscosity methyl-cellulose solutions may be substituted.

Implants and prostheses

Plastic, and even gold, implants have caused conjunctivitis. Plastic prostheses and even plastic contact lenses have resulted in allergic reactions.

*Cosmetics of importance in ophthalmology**

If we consider how universally cosmetics are used, the occurrence of allergies to cosmetics is infrequent, although many reports of such allergies are available.

Nail polish and nail lacquer. Of all ocular allergies traced to cosmetics, those due to nail polish and nail lacquer are by far the most common. Dermatitis involved the eyelids in 78 of 100 cases of all types of nail polish sensitivity in one reported series.[10] The offending ingredients are believed to be the synthetic resins in conjunction with the nitrocellulose.

Face powders. Face powders used to be sensitizers because of the orris-root and the rice-powder content. These ingredients are rarely used at present, having been replaced by titanium salts. Now any allergy to it is usually traced to perfumes, to pigments, and to impurities in manufacture and packaging.

Facial creams. Facial creams such as plain cold cream and vanishing creams are not often sensitizers. In both, the major offender is either lanolin or cocoa butter.

Lipstick. Lipstick dermatitis is usually due to the indelible dyes used, with failure to remove all traces of intermediates used in their manufacture.

Hair preparations. Hair dyes and rinses contain both sensitizers and toxic products. The most commonly used ingredient, paraphenylenediamine, may be both toxic and allergenic. Generally allergies occur from its use, but in susceptible persons serious toxic reactions may develop, sometimes terminating fatally. Paraphenylenediamine is also the major sensitizer in allergy to furs.

Eyelash dyes and mascara. Eyelash dyes, other than those of vegetable origin, are prohibited by public health authorities because of their paraphenylenediamine content. Allergies are infrequent. Allergy to mascara, when it occurs, is due to the base used, as the dyes employed, such as carbon black and methylene blue, are not sensitizers.

CHRONIC ALLERGIC CONJUNCTIVITIS

In contradistinction to the often dramatic, and generally severe, reactions that occur in the acute types of allergic conjunctivitis, the chronic forms frequently show little or no objective evidence of inflammation in comparison to the irritating subjective symptoms of itching, burning, and dryness that are usually present. As a result, the patients are often considered psychoneurotics, or else thought to be suffering from chronic catarrhal conjunctivitis.

Since many cases of chronic conjunctivitis are found bacteriologically negative, it may very well be that a significant number of such so-called cases actually are overlooked forms of chronic allergic conjunctivitis. In fact, even when the ophthalmologist is alerted to the frequency of chronic allergic conjunctivitis, if he performs routine epithelial scrapings in conjunctivitis he is often surprised to find numerous eosinophils in cases that clinically did not appear allergic.

The allergens responsible for chronic allergic conjunctivitis appear to fall into two main groups: (1) The usual exogenous airborne pollens, inhalants, and animal danders, as well as, sometimes, foods; and (2) bacterial products, of which staphylococcus toxin is the only one of importance.

The conjunctivitis that occurs often does

* In diagnostic testing for cosmetic sensitivity, instead of doing a patch test, it is better to apply the cosmetic daily in the way it is generally used, because in normal use the uncovered cosmetic loses much of its substance by evaporation. Covering the cosmetic by a patch does not permit this evaporation, and thus may give false-positive reactions with cosmetics that are actually harmless when used in the ordinary manner.[11]

TABLE 2

CHRONIC ALLERGIC CONJUNCTIVITIS

	Atopic Type	Bacterial Type
History:	Familial allergy frequent Other associated allergies	Familial allergy infrequent No associated allergies
Allergens:	Pollens, danders inhalants, foods	Bacterial products, mainly staphylococcus toxin
Allergic response:	Immediate	Delayed, bacterial type
Symptoms:	Burning, irritation and often severe itching	Burning, irritation and often marked dryness
Discharge:	May be mucopurulent	Watery, no pus
Lid margins:	Usually uninvolved	May be swollen; possible blepharitis
Conjunctiva:	May appear same in both types Often little objectively noted Sometimes edematous	May appear same in both types Papillary, but scattered follicles may appear. Dry, no chemosis
Cornea:	Generally negative	May have superficial punctate keratitis involving inferior portion of cornea
Epithelial scrapings:	Eosinophils almost always sooner or later found	No eosinophils
Cultures:	Normal flora	Toxin-producing staphylococci found, or else normal flora
Intracutaneous staphylococcus toxin or toxoid:	Moderate delayed response	Marked delayed response at least 3×3 cm., usually more. May have marked immediate response also. May get systemic reactions (arthritis)
Response to vasoconstrictors:	Local use often affords considerable symptomatic relief	Generally no great relief
Response to antihistaminics:	Oral use often affords marked symptomatic relief	Generally no relief at all
Response to cortisone:	Local use usually affords marked symptomatic relief	Relief in some cases, but not the rule
Best treatment:	Elimination of cause	Staphylococcus toxin or toxoid injections

not permit any ready means of distinguishing between the two types. One generally finds a low-grade recurrent irritative inflammation, in which the patient's major complaints may only be itching and dryness, and the objective findings limited to slight injection and perhaps, a tendency to folliculosis.

Yet, even with such overlapping, the appearance of the conjunctivitis, along with the history, the laboratory findings, such as epithelial scrapings, the clinical course, and response to therapy, all, when considered compositely, afford the framework of a practical means of differentiating the two types. In Table 2 an attempt is made to illustrate the differential diagnosis of the major forms of chronic allergic conjunctivitis. The table, which is offered on a tentative basis and will, undoubtedly, require future modification, should be read in conjunction with the text.

ATOPIC TYPE OF CHRONIC ALLERGIC CONJUNCTIVITIS

From the discussion just given and Table 2, it will be seen that in atopic individuals (persons with an allergic heredity) inhalants, pollens, danders, and foods can result in varying chronic forms of chronic allergic conjunctivitis. While the conjunctival reaction may range from microscopic papillary hypertrophy to moderate chemosis, the basic design of the case, (1) allergic background, (2) immediate type of allergic response, (3) eosinophils on epithelial scrapings, (4) great symptomatic relief with vasoconstrictors, antihistaminics, and cortisone, and (5) cure on elimination of the cause, differentiates it sharply from the bacterial type of chronic allergic conjunctivitis, and offers a practical clinical approach to the treatment of the patient.

There are two additional subgroups of chronic allergic conjunctivitis that should be classified among the atopic types.

Chronic edematous allergic conjunctivitis

According to Beigelman[20] this type has not been hitherto described. The conjunctival changes are mainly a marked edema of the lower transitional fold and the lower bulbar conjunctiva, which may later become relatively hard, pale, and glassy. The changes persist throughout the year, although the symptoms, itching and lacrimation, may be aggravated in warm weather. The secretion although scanty contains many eosinophiles.

Beigelman has examined six cases pathologically and finds mainly capillary engorgement edema and infiltration with small lymphocytes, plasma cells, and eosinophils. He cites the resemblance to nasal allergic changes, which were also found in his cases.

Similar boggy conjunctival changes, not so edematous, are often encountered in sufferers from chronic inhalant allergies, usually in individuals suffering from hay fever or asthma. The condition appears to me to be the conjunctival prototype of the nonspecific vasomotor rhinitis so frequently noted in hay-fever sufferers after the season of pollenation ends, and in many other types of patients.

Chronic simple allergic conjunctivitis

Bourquin[21] has described a recurrent bilateral simple allergic conjunctivitis occurring in Geneva, Switzerland, during the warm season. Often only a slight hyperemia of the transparent conjunctiva is present, with fine papillae visible only with the slit-lamp. A small preauricular lymphadenopathy is the rule. The absence of bacteria and the presence of conjunctival eosinophilia, along with slight blood eosinophilia, mark the condition as allergic.

Histologic examination reveals epithelial and subepithelial edema, with greatly dilated capillaries and a cellular infiltration in which eosinophils predominate, both in the tissue and blood vessels; plasma cells and polys are present, as are basophils.

Although the condition resembles early vernal conjunctivitis, and, indeed, occurs at the same time of the year, no matter how long it persists, it never leads to fibrous interstitial changes like vernal conjunctivitis does. Bourquin's cases would seem to be due to pollens.

BACTERIAL TYPE OF CHRONIC ALLERGIC CONJUNCTIVITIS

The contributions of Woods[22] and of Burky[23] have established that hypersensitivity to staphylococcus toxin is the cause of many cases of chronic recurrent conjunctivitis. Clinically these cases, as described by Woods, are characterized by dryness and absence of purulent discharge, folliculosis, marginal blepharitis, reddened swollen margins of the eyelids, frequently slight secondary corneal changes, the isolation of a toxin-producing staphylococcus from the conjunctiva, and the presence of a high degree of cutaneous sensitivity to staphylococcus toxin.

Sometimes, even when the other criteria for diagnosis are all present, cultures show no staphylococci on repeated tests, these having been eradicated but leaving the local hypersensitivity. Only desensitization by means of injections of staphylococcus toxin or toxoid, effects a cure.

Woods feels that the mechanism of the conjunctivitis is: first, staphylococcal infection resulting in tissue hypersensitivity. Subsequent fresh infections, even if the original infecting organism is eliminated, results in recurrences of the conjunctivitis. He considers that a reaction of more than 3 by 3 cm. to an intradermal injection of 0.1 cc. of a 1:100 dilution of staphylococcal toxin is evidence of such hypersensitivity. He has found the toxin not to be irritative to the normal skin or mucous membrane.

On the other hand, Allen[24,25] has produced conjunctivitis experimentally with the use of staphylococcus toxin in persons pre-

sumably not hypersensitive to the staphylococcus. Perhaps a quantitative factor enters into the problem.

The importance of toxin-producing staphylococci in the causation of blepharoconjunctivitis has been shown by the work of Thygeson.[26] In these cases obvious ulcerative blepharitis is associated with papillary conjunctivitis and a rather diagnostic superficial keratitis of a toxic origin. Meibomitis is often present. Cultures of the lid margins and conjunctiva show many staphylococci. Epithelial scrapings of the lid margins often show polymorphonuclear leukocytes and staphylococci. Treatment, in addition to the eradication of the bacteria, often requires the use of vaccines or toxoid.

If one could rule out bacterial allergy as a factor in blepharoconjunctivitis, the success of vaccine therapy might be attributed to active immunization. However, even in this condition we encounter numerous patients with a high degree of skin sensitivity to staphylococcus toxoid or vaccines.

Some allergists maintain that positive skin reactions to bacterial filtrates of themselves are not diagnostic. However, a recent experience of mine would indicate that bacterial allergy may unquestionably develop in active long-standing staphylococcal infection.

This experience concerned a patient with a most severe infectious eczematoid staphylococcal dermatitis of the eyelids, of 10 years' duration, on whose conjunctivas and lid margins were found innumerable colonies of toxin-producing, hemolytic, coagulase-positive Staphylococcus aureus.

Intradermal injections of autogenous vaccine and staphylococcus toxoid gave marked immediate reactions. Even injections of minimal doses had to be discontinued because on several occasions the patient developed transient acute arthritis of the fingers. Symptoms due to chronic cholecystitis also became aggravated following these injections.

There could hardly be any question that in this patient a most exquisite allergy to the staphylococcus and its products was present. Incidentally, the use of silver nitrate was the major element in effecting a most dramatic improvement.

The importance of Woods' contribution is particularly appreciated in those cases of chronic conjunctivitis in which diagnostic tests are essentially negative except for a high degree of skin sensitivity to staphylococcus toxin or toxoid. These patients often show few, if any, bacteria on culture of the conjunctiva and not very many more on the lid margins. They often have periods of exacerbation of their complaints with some objective increase in the conjunctivitis. Many do extremely well with staphylococcus-toxoid injections.

Further evidence in favor of Woods' concept may be obtained from patients with chronic conjunctivitis associated with infection of the paranasal sinuses by toxin-producing staphylococci. Conjunctival cultures are generally negative; skin tests may be definitely positive.

The conjunctivitis often responds to injections of vaccines and toxoid, or to treatment of the exciting focus by either intensive antibacterial therapy or surgery. I use toxoid because it is readily available commercially. Toxoid differs from toxin first in that it has been detoxified with formaldehyde, and also in that it appears to have additional hemolyzing and dermonecrotizing factors.

It is my feeling that we should interpret these reactions as we do those of the tuberculin test. A strongly positive reaction to a weak concentration indicates marked sensitivity and is diagnostically suggestive, a mildly positive reaction merely indicates prior infection at some time or other in the past, unless the reaction is recently acquired.

The patients who exhibit the greatest skin sensitivity generally are those with clinical evidence of low resistance to toxic staphylococci, as evidenced by marked exacerbations when reinfection occurs.

TABLE 3

CAUSES OF CONJUNCTIVAL EOSINOPHILIA

1. Vernal conjunctivitis (also basophilia)
2. Allergic conjunctivitis (also basophilia, especially in drug allergies)
3. Conjunctivitis due to various chemical and vegetable irritants; (lime, turpentine, indelible pencil, insect powder, ipecac, ricin, etc.)
4. Conjunctivitis due to parasites, myiasis and insects, hornet-sting, sporotrichosis and trypanosomiasis
5. Ocular pemphigus (low-grade eosinophilia)
6. Rarely in bacterial and viral conjunctivitis (since bacterial allergy does not cause conjunctival eosinophilia, in these instances infection may be superimposed on allergy)

CONJUNCTIVAL EOSINOPHILIA

The finding of eosinophils on conjunctival smears or scrapings is almost pathognomonic of allergic conjunctivitis or vernal catarrh, assuming that we are able to rule out other causes of conjunctival eosinophilia. A positive finding is diagnostically valuable; the failure to find any, however, does not rule out allergy. It is generally easy on clinical grounds to differentiate nonallergic conjunctival eosinophilia from the allergic types. Table 3 lists the major causes of conjunctival eosinophilia and basophilia.[18]

There are a number of points concerning conjunctival eosinophilia that should be stressed. Eosinophils may not appear for some time, perhaps two weeks after the allergy has manifested itself. This is especially so in drug and cosmetic allergies, but can even occur in vernal catarrh.

As already mentioned, irritation to pilocarpine, eserine, DFP, and other miotics does not cause eosinophilia or basophilia. Neither does it occur in bacterial types of allergy. Furthermore, the day-to-day variability of the number of eosinophils found, even in allergies of long standing, is notorious, so that it is dangerous to assume that a conjunctivitis is improving merely because fewer eosinophils have been found on that occasion.

There is no correlation between blood eosinophilia and the presence of eosinophils in the conjunctiva. Even in the presence of blood eosinophilia of very high degree, scrapings of the normal conjunctiva will show no eosinophils unless bleeding occurs.

Lastly, despite the role that histamine plays in allergic reactions and despite the frequency that eosinophils are found in allergies, there is no connection between the two. Eosinophils do not contain histamine.

TREATMENT OF ALLERGIC CONJUNCTIVITIS

Prerequisites for the successful management of cases of allergic conjunctivitis are: (1) Recognition, first, that an allergy is present; (2) the proper classification of the type of allergic conjunctivitis present; and (3) the discovery, if possible, of the sensitizing substance.

If one proceeds in this manner, considerable time, as well as expense to the patient, will be saved because it is often not necessary to determine the exact cause of the allergy in order to afford the patient relief. The important thing is to have a well-organized clinical approach to the subject, based on an understanding of the mechanisms involved and the types of allergic conjunctivitis that may occur. In this way, the treatment of many cases will prove relatively easy, responding to nonspecific measures and drugs.

Even in those cases that present difficulties, the proper clinical understanding of this group of conditions will prove more valuable than blind reliance on sensitivity tests performed by the allergist. These, of course, are often necessary and sometimes vital to successful therapy, but do require clinical evaluation as a governing factor.

ELIMINATION OF THE CAUSE

The most effective therapeutic measure in allergic conjunctivitis is to eliminate the cause by either removing the offending antigen if possible or having the patient avoid it. This is by far the best treatment, but it depends, first, on arriving at a more or less

definite etiologic diagnosis, and, second, on the feasibility of such a separation. In cases of bacterial allergy it is of course entirely impossible to do the latter, and often economic and other factors prevent such a procedure in air-borne and contact allergies. As far as the diagnosis is concerned, even if allergic studies are not helpful, often enough routine detective work finally reveals the allergen.

Contact allergy to either drugs or cosmetics is generally the easiest to eliminate, unless the use of the drug is vital. As regards cosmetics, even if the usual hypoallergic preparations are ineffective, it is not necessary to condemn the unfortunate lady to a bleak and pale existence. The manufacturers are ready and willing to make up special products for such unhappy individuals.

Specific desensitization

Except in pollenosis, specific desensitization in nonbacterial allergy does not appear to be of great benefit in allergic conjunctivitis. Moreover, the improvement, when it does occur, often is only temporary. The management of desensitization is reserved for the allergist, and in general should not be advised if other measures are sufficiently successful.

In the bacterial type of allergic conjunctivitis, elimination of the offending bacteria is impossible and adjuvant treatment is not very effective. Woods suggests desensitization with staphylococcus toxin using 0.1 cc. intradermal injections of a dilution of toxin that does not cause a large initial reaction. He usually starts with 1:100 dilution, but in more sensitive individuals uses the 1:1,000 strength. The same process is repeated with the next stronger concentration until it too causes no reaction. When a patient is able to tolerate 0.1 cc. of a 1:10 dilution this dose is maintained by weekly injections for six months to maintain desensitization and prevent recurrence of the sensitivity.

I have obtained good results in cases that appeared to be due to bacterial allergy to the staphylococcus, with the use of commercially prepared staphylococcus toxoid. My regime has been basically the same as in the treatment of staphylococcal blepharoconjunctivitis. Indeed, the mechanisms involved may be similar.

An initial 0.1 cc. intradermal injection of diluted toxoid (1:10) serves as the diagnostic and therapeutic basis. While reactions to various commercial brands vary and it is therefore advisable to use one product constantly, any reaction larger than 1.0 by 1.0 inch is considered suggestively positive, although I hesitate to place too much reliance solely on the skin reaction.

Succeeding injections are made intradermally once or twice a week and increased slowly, depending to a large extent, but not entirely, on the skin reaction. When a dose of 0.2 cc. of the original toxoid is reached, and thereafter, 0.1 cc. of the dose is injected intradermally and the remainder then introduced subcutaneously. When the dose of 1.0 cc. is attained, injections are continued at this strength, but at decreasing intervals, first every two weeks, then every month for six months.

Drug therapy

A number of new types of medicaments, as well as older drugs, are valuable in the treatment of allergic conjunctivitis.

Antihistaminics. The theoretical basis of antihistamine therapy is that histamine plays a prominent part in anaphylaxis and allergy, and that the antihistaminics nullify its action by combining with the target cell, preventing the absorption of histamine by these cells and, thus, the resultant allergic reaction.

There is no question that the oral administration of antihistaminics is often of great value in seasonal hay fever and, therefore, is valuable in allergic conjunctivitis due to it. There is considerable doubt on both theoretical and empirical grounds, however, that antihistaminics are of any use in the delayed type of allergic response. Neither the Arthus

phenomenon nor the bacterial type of sensitivity appear to be influenced by the drugs.

Nevertheless, some investigators have reported beneficial results in such types of allergic conjunctivitis especially to drug and other contact allergies. It has been claimed, moreover, that in some cases the combined use of antihistaminics both orally and topically has permitted the continued use of the sensitizing medicaments. My own experience has not been so fortunate.

While the oral administration of antihistaminics in the type of reaction to an inhalantlike perfume may alleviate the nasal symptoms, the eyes continue to itch anyway. In marked drug allergies I have been unimpressed with the use of antihistaminics in any form. Local use does not seem effective in most instances.

In this connection, it should be stressed that the entire group of drugs appears to act as mild local anesthetics, which would complicate the interpretation of any beneficial results. Antistine stings so much that patients do not like it; the results obtained do not justify its use in most cases. Chlortrimeton locally does not cause such irritation and may be more effective.

Cortisone and ACTH. Recent experimental studies have confirmed the clinical observations that both of these agents have profound effects on allergic reactions. Woods[27] has shown that both ACTH, administered systemically, and cortisone, administered systemically or topically, have a distinct temporary inhibitory effect on the inflammatory and exudative phases of protein, drug, and bacterial sensitivities but do not appear to have any effect on the underlying allergic state. Cortisone and ACTH also appear to block other types of ocular inflammation as well, making cells temporarily immune to the effects of bacteria and chemical agents, as well as allergens.

Clinical experiences with these drugs indicate their great value in allergic diseases[28] and ophthalmologists, by now, are all aware of their value in allergic conjunctivitis and related conditions. In drug allergies the injection of ACTH has allowed the continued use of the offending drug.

A critical level of dosage must be maintained however. In a case of atropine allergy reported by Carey et al.[29] 40 mg. of ACTH daily allowed the continued use of atropine, but 20 mg. per day did not. During treatment, a patch test, positive before and afterward, was negative.

The use of topical cortisone in some cases does permit similar continued use of a sensitizing drug but in other cases does not. Here too it appears to be a matter of a critical dosage level in relation to the degree of sensitivity that is present. If the offending medicament is first stopped and the conjunctivitis is allowed to subside, pretreatment with topical cortisone for at least 24 hours should permit the use of weak solutions of the allergenic drug as long as cortisone is continued, in many instances.

Hydrocortisone sometimes is effective upon local use in instances where cortisone has been disappointing.

Because ACTH and cortisone appear to block other types of inflammatory reactions besides those due to allergies, one should not assume that just because cortisone relieves an ocular inflammation the condition is allergic. However, as the leavening influence of time and further clinical experience begins to make itself felt, it does appear more and more that cortisone's greatest value is in inflammations with, at least, an allergic component. We do know now that in certain virus diseases the drugs appear not to be effective and may even be harmful.

Other medicaments. Other palliative remedies include local anesthetics, vasoconstrictors, and antiseptics all often used together.

Anesthetics. Cocaine still is a favorite because of its relatively low index of allergenicity. It may be used in rather weak solutions, 0.25 to 0.5 percent. Holocaine is also valuable. Pontocaine and others are more likely to cause sensitivities.

Vasoconstrictors. Epinephrine, privine,

ephedrine, and neosynephrine still have a very important place in the treatment of allergic conjunctivitis. In fact, they allay the acute symptoms of pollen allergies much more effectively and rapidly than any other local medicaments.

Antiseptics. Certain ones, notably those fatty acid derivatives which do not cause irritation or allergy, such as sodium propionate,[30,31] often are useful in that they counteract secondary infection and also have some soothing effects. They may be safely used in bacterial infections occurring in allergic individuals in whom the antibiotics, sulfonamides, or mercurial antiseptics may result in new sensitizations.

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ELECTRO-ENCEPHALOGRAM AND OCULAR FUNCTION*

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This report concerns a study of eye-muscle function, visual acuity, fusion, stereopsis, and ocular dominance in 50 children on whom the electro-encephalogram was made routinely. The criteria by which the children were selected for this examination were: (1) Their I.Q.s were above 85, and (2) they were dyslexic.[1] Cases which had a history of brain injury were not accepted.

The review of these cases was stimulated by a paper written by Levison, Gibbs, Stillerman, and Perlstein, "Electro-encephalogram and eye disorders."[2] Their conclusions were:

"Thirty percent of 36 children with strabismus but otherwise normal, and only 0.5 percent of 180 children entirely normal, had occipital abnormalities in their electro-encephalograms.

"In children with organic brain disease causing cerebral palsy, electro-encephalographic foci in the occipital region occurred almost twice as frequently in the presence of abnormal eye findings. The relationship prevailed regardless of the presence or absence of seizures.

"In patients with eye disorders, electro-encephalographic foci involved the occipital region six times more commonly than all other foci combined."

For the purpose of this study the electro-encephalographic findings are classified, for convenience, into three types: Type 1, normals; Type 2, amplitude asymmetry in the occipital areas; Type 3, disturbance in the temperoparietal areas plus positive spikes in the hemispheres and slow activity.

The distribution, percentagewise, of the cases in this classification was Type 1, 72 percent; Type 2, 18 percent; Type 3, 10 percent.

To determine whether a correlation existed between duction abilities and the electro-encephalographic findings, a detailed analysis was made (table 1). It was felt that by this means, a more accurate interpretation could be made of these relationships if done before gross changes had occurred in the neuromuscular function as evidenced in strabismus. During the measurements of ductions, care was taken with each individual to obtain accurate reports as to the point at which the image separated and diplopia appeared and the point of recovery—where the two images fused again. Arbitrary standards and their limits were selected to signify low ability, normal, and above normal ability.

The data in Table 1 indicate that, even with a relatively low prism strength considered as "normal" in measuring the diplopia and recovery point, weak fusion amplitude, with or without electro-encephalographic abnormalities, is definitely associated with dyslexia, especially weak recovery from diplopia.

It is evident from Table 1 that the overall ratio of performance abilities in the lower ability group to the normal group shows no significant changes in the abilities concurrent with the various electro-encephalographic abnormalities.

* From the Department of Ophthalmology, Northwestern University, and the Dyslexia Memorial Institute.

TABLE 1

ANALYSIS OF DUCTIONS
(Percentage of entire series)

	Diplopia considered Normal limits	Recovery from diplopia considered Normal limits	Low Ability			"Normal" Ability			More than Normal Ability			Total (percent)		
			Type 1‡	Type 2	Type 3	Type 1	Type 2	Type 3	Type 1	Type 2	Type 3	Low	Normal	High
DISTANCE														
Adduction	22–14*		20	8	0	40	8	10	12	2	0	28	58	14
Adduction		14–6*	26	8	4	42	10	6	6	0	0	38	56	6
Abduction	12–5†		32	6	6	36	12	6	4	0	0	42	54	4
Abduction		7–2†	20	8	2	48	10	8	4	0	0	30	66	4
NEAR														
Adduction	32–20*		16	6	0	42	6	6	14	6	4	22	54	24
Adduction		16–9*	22	6	0	38	6	8	12	6	2	28	52	20
Abduction	18–10†		14	8	4	42	8	2	16	2	4	26	52	22
Abduction		9–3†	14	10	2	46	6	4	12	2	4	26	56	18

* Prism base-out.
† Prism base-in.
‡ Remember that Type 1 is normal electro-encephalogram; Type 2 is occipital asymmetry; Type 3 is abnormal electro-encephalogram in other areas.

Evidently, when there is no history of injury and before gross changes occur, as in strabismus, the occipital asymmetry areas are of no greater significance in causing low duction abilities than other disturbances as determined by the electro-encephalogram—or even in normal electro-encephalograms. The incidence of occipital asymmetry in the lower ability group was relatively the same as in the "normal" group.

It was felt that when the fusion impulse was eliminated and the relative positions of the eyes were measured while in a position of rest, some clue could be found to determine the influence of the electro-encephalograms on the neuromuscular mechanism of the eyes—if there were any demonstrable influence.

As a standard for measuring phorias, from four degrees esophoria to four degrees exophoria was considered as orthophoria: less than two degrees was normal for hyperphoria. Only four percent had hyperphoria. Our analysis is made in Table 2.

TABLE 2

ANALYSIS OF PHORIAS
(Percentage of entire series)

	Type 1	Type 2	Type 3	Total
Orthophoria	46	10	6	62
Esophoria	12	6	0	18
Exophoria	14	2	4	20

One observes that the ratios of muscular imbalance in the various classifications of the electro-encephalogram were relatively the same. It is questionable whether abnormal electro-encephalograms have any significant independent association with the peripheral ocular muscle mechanism, even when the fusion impulse is eliminated.

Thus far attention has been directed mainly to the motor aspects of the peripheral ocular muscular mechanism. In order to determine the integrity of the neuro-optic pathways and visual cortex and their relationship to electro-encephalographic findings, a further investigation was made. If there was a disturbance in the associational pathways, there should be evidence of defective interpretations of visual acuity, fusion, and stereopsis.

The findings dealing with visual acuity in this series of cases are given in Table

TABLE 3

ANALYSIS OF VISUAL ACUITY
(Percentage of entire series)

	Type 1	Type 2	Type 3	Total
Normal Vision	56	14	10	80
Subnormal in one eye only	2	2	0	4
Subnormal in both eyes	14	2	0	16
Amyblyopia	0	0	0	0

3. These data were tabulated before a refraction was done.

In all cases refraction was performed under a mydriatic and the visual acuity was improved to normal. Evidently, these types of abnormal electro-encephalograms had no significance in relation to visual acuity in these patients.

Furthermore, the report of Levison, Gibbs, Stillerman, and Perlstein did not show that fields of vision had been taken on their cases. Neither were the fields taken in our series. This procedure should definitely be instituted before accepting the premise that the occipital cortex is pathologically involved.

The exact process by which fusion is accomplished is not known. It can be safely assumed though that, if there is a marked disturbance in the associational pathways, there should be faulty fusion and thereby a hindrance to the acquisition of stereopsis, as well. An analysis of the findings is given in Table 4.

Since fusion was present in practically all cases (96 percent) fusion per se is not greatly influenced. Furthermore, the relatively high incidence of the absence of stereopsis should not be interpreted as due to organic lesions of the associated pathways. As fusion and stereopsis are acquired oculocerebral processes, in clinical experience stereopsis can be learned after fusion is attained by orthoptic training, almost without exception.

The visual process is complex and is affected not only by the physical state of the peripheral optic mechanism but also by the central perceptual and conceptual mechanism concerned in fixation, which may in turn affect the peripheral optic mechanism through the autonomic as well as the skeletal nervous system.

TABLE 4

ANALYSIS OF FUSION AND STEREOPSIS

	Type 1	Type 2	Type 3	Total (percent)
FUSION				
Present	70	16	10	96
Absent	2	2	0	4
STEREOPSIS				
Present	58	14	10	82
Absent	14	4	0	18

TABLE 5

DETERMINATION OF OCULAR DOMINANCE

	Type 1	Type 2	Type 3	Total (percent)
Perfect projection	54	12	18	74
Simultaneous binocular vision but not perfect projection	4	0	2	6
Simultaneous binocular vision but suppresses one eye at times	4	2	0	6
Alternates	4	0	0	4
Suppresses one eye completely	6	4	0	10

An important factor in the perceptual and conceptual processes is the ability to project the images of both eyes simultaneously into space at corresponding positions, thereby aiding the individual to orient himself properly to his surroundings.

The main functions of the visual mechanism would seem to be an integration of associated movements, which vary among themselves according to the stimuli which exists at any particular time and influence the sensorimotor system of the whole conscious visual process. These adjustments are only relatively steady or fixed in the homeostatic sense. The degree of their stability depends on the relative degree of normality of all the diverse activities which are synthesized into the whole conscious visual process.

If one eye is dominant, then, with the elimination of the fusion impulse, there would be confusion in the projection processes unless the nondominant eye was projecting its image to the identical point in space or unless its image was suppressed.

In checking for ocular dominance and its relationship to abnormal encephalographic findings, a stereoscope was used. By using

dissimilar cards that stimulated fusion only slightly, the probability of introducing an extraneous factor in determining the dominance was eliminated. For, as soon as an extraneous act such as pointing and sighting, is used, it becomes expedient to choose one eye or the other as the dominant eye, even though both eyes are maintaining fixation and fusion of the object observed.

An analysis of the findings observed in this procedure is made in Table 5.

Under the condition of these measurements it would seem that, if the abnormal electro-encephalographic findings influenced either the perceptual processes or the peripheral ocular mechanism in their adjustments and interpretations, it would become evident. Even in this triggerlike adjustment, involving the conscious visual processes, there are no significant differences between the abilities to have perfect projection in abnormal or normal encephalograms.

Summary and Conclusions

Fifty routine electro-encephalograms were made on routinely selected children. Cases with history of brain injury were avoided. The criteria in the selection were: I.Q. above 85, and dyslexia.

The electro-encephalographic abnormalities for this study were classified for convenience into Type 1, normal activity; Type 2, amplitude asymmetry in the occipital areas; and Type 3, disturbance in the temperoparietal areas, positive spikes in the right and left hemispheres, and slow activity. The incidence was: Type 1, 72 percent; Type 2, 18 percent; Type 3, 10 percent.

A detailed analysis is made of ductions. Arbitrary standards and their limits were selected to signify low duction ability, "normal," and more than normal ability. It is evident that the over-all ratio of performance abilities to the normal shows no significant changes in the electro-encephalographic abnormalities.

The ratio of muscular imbalances in phorias was relatively the same for the three types of electro-encephalograms.

There was no change in visual acuity either in the cases with normal or abnormal encephalograms.

There was no significant correlation between abnormal and normal brain waves and fusion ability.

Stereopsis also showed practically the same ratios between normal and abnormal encephalograms.

In determining ocular dominance and abilities to project the images properly there was no significant difference between abnormal and normal cases.

The incidence of abnormal electro-encephalographic foci observed by Levison, Gibbs, Stillerman, and Perlstein in "normal" and "normal with strabismus" cases was 32 percent compared to 28 percent in our series of routinely selected cases.

The ratio in our series was: practically, occipital disturbances were to other focal abnormalities as 2:1; while in their cases the ratio was 30:2.

They concluded that because such a high degree of occipital asymmetry occurred it must be an abnormal finding since it did not occur in their normal control group; yet in our group which can be considered a control group, since brain injury cases were not involved, the sum of the abnormalities equaled their total.

Electro-encephalographic findings should be considered merely an adjunct, as any other laboratory procedure, in making a diagnosis. One must rely mainly on the history and clinical findings before making a diagnosis.

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INCREASED INTRAOCULAR PRESSURE PRODUCED BY DI-ISOPROPYL FLUOROPHOSPHATE (DFP)*

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Since the first report in the ophthalmic literature by Leopold and Comroe,[1,2] on the use of di-isopropyl fluorophosphate (DFP) in the treatment of glaucoma, this substance has become commercially available and its use has become relatively widespread. All of the reports since the original one, with the exception of that by Marr,[3] range from mildly to very enthusiastic.

Despite this enthusiasm, most of these reports also mention the disadvantages of DFP, which include discomfort or actual pain following its instillation, particularly when the patient first starts using the drug; blurring of vision due to the severe ciliary spasm which it causes; and paradoxical increase in intraocular pressure. It is with the last-named disadvantage—the paradoxical increase in pressure—that this report is particularly concerned.

REVIEW OF THE LITERATURE

McDonald[4] discussed the paradoxical rise in intraocular pressure in his report on the use of DFP in eyes which could not be controlled by other miotics. He found an initial rise of pressure of 3.0 to 7.0 mm. Hg in seven patients but subsequently, after 48 hours, all seven showed an appreciable drop below the original level. In 10 patients, there was an initial rise of from 2.0 to 35 mm. Hg, and in only one did the pressure fall below the original level after 48 hours.

Three uncontrolled patients who required surgery were made worse by the use of DFP.

One patient was admitted for surgery of the left eye and DFP was instilled in both eyes; in spite of adequate miosis, the pressure of both eyes started to climb, reaching 48 mm. Hg, and was relieved only by surgery.

A second patient with iris atrophy in the right eye was placed on DFP; the pressure, which had been in the 20's with another miotic, rose to 48 mm. Hg where it stayed, despite return to the original miotic.

McDonald stated that DFP might hasten rather than retard the need for surgical intervention, and felt that arteriolar vasodilatation and/or ciliary spasm caused by the drug might swing the pendulum in the wrong direction. He advocated checking the intraocular pressure 45 to 60 minutes following the instillation of DFP and, if there is an initial rise, watching the patient very closely until the pressure drops to the original level or below.

Marr reported on 32 eyes uncontrolled by other miotics in which only five were controlled by DFP and mentioned the fact that an occasional increase in pressure may occur. While he did not cite any specific cases, he did say that in three out of four eyes with "congestive glaucoma" in which DFP was used, there was a shallowing of the anterior chamber without, however, any increase in pressure. He also noted a reduction in depth of the anterior chamber in "several" eyes with chronic glaucoma simplex.

Leopold and McDonald[5] felt that DFP produced better results in narrow-angle glaucoma than in wide-angle glaucoma. In 370 eyes which they studied, they found six which responded to DFP by an increase in intraocular pressure.

They felt that either one or both of two mechanisms might be capable of causing such a response: (1) Engorgement of the ciliary body as the result of increased capillary permeability, resulting in a me-

* From The Michael Reese Hospital. Presented before the Chicago Ophthalmological Society, December, 1952.

chanical narrowing of the chamber angle; or (2) increased protein content of the aqueous which might also interfere with the drainage mechanism. They suggested that the simultaneous use of a vasoconstrictor as proposed by Sugar or of an antihistamine as proposed by von Sallmann might prevent any rise in pressure.

Hughes,[6] in discussing this paper by Leopold and McDonald, stated that results in 40 eyes treated with DFP were not encouraging and that three eyes in the series developed an acute rise in tension. Two eyes of one patient which he mentioned briefly in his discussion are included in this report (Case 1).

Haas[7] described several cases which were favorably influenced by DFP and one case which was made worse.

This case was that of a 72-year-old woman who had glaucoma with normal fields and vision of 20/20. Gonioscopically there was a narrow-angle entrance. Her intraocular pressure was well controlled with pilocarpine six times daily. When placed on DFP once daily, her pressure rose from between 21 and 33 to 45 mm. Hg, even though there was maximal miosis. The use of pilocarpine was resumed and the pressure gradually fell to between 27 and 31 mm. Hg.

Haas postulated that DFP in this instance produced further narrowing of an already narrow angle, resulting in a mechanical blocking. As a result of his experience, he advocated the use of DFP in aphakic glaucoma but cautioned against its use in narrow angle glaucoma. This is advice which he has recently reiterated.[8] At the present time, he feels that DFP should be used only in aphakic glaucoma.

Raiford[9] reported on the use of DFP in 42 eyes in 24 patients. In one patient of this series, pressure went up to 56 mm. Hg after five days of therapy but promptly dropped to an average of 21 mm. Hg and was well maintained after stopping the DFP and changing to five-percent pilocarpine.

Ferrer[10] mentioned that one patient in a series of 31 eyes in 17 patients treated with DFP developed an increased intraocular pressure.

Bond[11] reviewed the literature on the treatment of glaucoma with DFP and reported on 30 cases which he had followed. He apparently had no cases in his series in which the pressure rose; however, he warned that this is not a drug that can be used lightly because it may cause a sudden rise in pressure instead of a fall as a result of vasodilatation and engorgement of the ciliary body, and that these changes might further embarrass an already embarrassed angle.

Shaffer,[12] in discussing Bond's paper, emphasized the same thing and said that DFP should be used only after carefully appraising the risk.

Hauck and Biggins[13] reported on 17 cases of glaucoma which were refractory to other forms of treatment and were placed on DFP; four of these cases responded satisfactorily at first but later showed an increase in intraocular pressure.

Kravitz[14] discussed his experience with 90 cases, mentioning two cases in which the pressure rose considerably. He advocated the use of phenylephrine with DFP and suggested the use of DFP in all cases not responding to other miotics.

Recently, Butler[15] reported the case of a 68-year-old man with a narrow angle in whom an acute attack of glaucoma was produced after the instillation of three drops of 0.1-percent DFP. Medical treatment failed to lower the pressure, which was 83+ in each eye, and a bilateral iridencleisis was done 24 hours after the acute rise in pressure, with ultimate satisfactory results.

A summary of cases is shown in Table 1. Those reports in which the number of cases or eyes was not specified are not included.

Case reports

Case 1

A. Z., a 64-year-old white man, was first seen by one of us (T. N. Z.) on September 10, 1941, complaining of impaired vision for distance and near and desiring a routine examination. The

TABLE 1

SUMMARY OF THE LITERATURE

Authors	Date	Total Eyes	Rise in Intraocular Pressure		
			Slight	Unspecified	Marked
Leopold & Comroe[1]	1946	40			
McDonald[4]	1946	122	7		10
Raiford[9]	1949	42			1
Stone[16]	1950	150	14		10
Leopold & McDonald[5]	1948	370		6	
Hughes[6]	1948	40			3
Haas[7]	1948	8			2
Kravitz[14]	1950	180			4
Ferrer[10]	1950	31	1		
Hauck & Biggins[13]	1949	34*	8*		
Scheie[17]	1949				1
Von Sallmann & Dillon[18]	1947	50	5		

* Original report does not specify whether this figure refers to the number of eyes or patients.

visual acuity on this date was 20/200 in each eye corrected to 20/25 in each eye, and ability to read J3 print at 18 inches.

External examination of the eyes revealed no significant abnormal findings. The anterior chambers were of moderate depth. The intraocular pressure as measured with a Gradle-Schiøtz tonometer was 20 mm. Hg in each eye.

Refraction performed under euphthalmine mydriasis and verified subsequently indicated the following refractive error:

R.E.: +2.0D. sph. ⊃ +1.5D. cyl. ax. 5°
L.E.: +2.0D. sph. ⊃ +1.5D. cyl. ax. 165°
Add +2.0D. sph., O.U., for near

This correction provided 20/15 vision in each eye for distance and ability to read J1 print at 14 to 20 inches. Examination of the ocular media and fundi at this time revealed no pertinent abnormality.

The patient was seen through the subsequent years for a periodic check-up of visual acuity and for routine refraction. On November 21, 1944, he still had a visual acuity of 20/20 in each eye, and the intraocular pressure again measured 26 mm. Hg in the right eye and 17 mm. Hg in the left eye with a Gradle-Schiøtz tonometer.

On March 16, 1945, the intraocular pressure was 24 mm. Hg in the right eye and 20 mm. Hg in the left eye.

On May 1, 1945 the patient was seen with complaints of cloudiness of vision in both eyes, especially at night, accompanied by haloes about electric lights. At this time the anterior chamber of each eye was shallow. There was a faint suggestion of a beginning temporal excavation visible in the right nerve head. The left optic nerve appeared flat and within normal limits. The intraocular pressure measured 28 mm. Hg in the right eye and 20 mm. Hg in the left eye.

A diagnosis of bilateral narrow-angle glaucoma was considered and therapy was instituted with pilocarpine (one percent) as eye drops in each eye two times daily. One week later the pressure was recorded as 24 mm. Hg in the right eye and 20 mm. Hg in the left eye.

For the next two years the patient continued on this routine of therapy. Periodic testing of the intraocular pressure revealed a range of 24 mm. Hg down to 13 mm. Hg in the right eye, and 20 mm. Hg down to 11 mm. Hg in the left eye. In spite of the pressure being seemingly controlled at this level, there were still periodic episodes of pain in either eye, at which time the patient would instill additional medication in each eye with subsequent relief.

On May 17, 1947, the corrected visual acuity was 20/20 in each eye. It was now noted, however, that the anterior chamber of each eye was extremely shallow. The pupils were contracted due to the miotic and the intraocular pressure was 42 mm. Hg in the right eye and 17 mm. Hg in the left eye.

This was the first significant elevation in pressure recorded up to that date, and pilocarpine (two percent) twice daily in each eye was recommended. This promptly reduced the pressure, and two weeks later the tension was 20 mm. Hg in the right eye and 14 mm. Hg in the left eye.

For the next seven months of 1947, the ocular tension seemingly remained well controlled and ranged from 20 to 26 mm. Hg in the right eye and 14 to 20 mm. Hg in the left eye. The patient was becoming lax in using his medication, however, and stated that he only used the miotic when pain appeared in each eye.

On March 24, 1948, there was an intraocular pressure of 50 mm. Hg in the right eye and 15 mm. Hg in the left eye, and the frequency of miotic instillation in the right eye was increased to four times daily. Two weeks later the intraocular pressure was measured at 15 mm. Hg in each eye. However, the patient stated that, during this interval, he had experienced two episodes of severe pain and blurred vision in the right eye. He admitted he had been remiss in using the miotic as directed and, with the onset of blurred vision and pain in the right eye, he instilled the miotic in the

eye at frequent intervals and thus avoided further complication.

In view of the negligent manner in which the patient had been using his miotic drops, it was considered advisable to try DFP in order to control more adequately the pressure with instillation of drops at less frequent intervals. With this in mind, on April 14, 1948, the patient was given a 0.05-percent solution of DFP in peanut oil with instructions to use one drop in each eye twice daily.

For the next three days this miotic was used as directed and on April 14, 1948, just four days later, after having used a total of six drops of DFP in each eye over a three-day period, the patient experienced an attack of excruciating pain in the left eye with marked impairment in vision in this eye, associated with a rainbow effect about electric lights. He was seen as an emergency in this crisis and the appearance of each eye was striking.

Both eyes were extremely pale white with no visible evidence of congestion. There was only a faint edema of the corneal epithelium of the left eye. The anterior chamber of each eye was not visible upon examination with the slitlamp and corneal microscope. The iris-lens diaphragm was lying against the posterior corneal surface. The pupil of each eye was pinpoint. The intraocular pressure of the right eye was 37 mm. Hg and of the left eye 82 mm. Hg, as measured with a Sklar-Schiøtz tonometer. The patient was admitted to Michael Reese Hospital for treatment of this unusual type of acute glaucomatous crisis.

The use of DFP was discontinued and treatment instituted with 0.25-percent eserine, two-percent pilocarpine, and 2.5-percent neosynephrine drops in each eye and 50 cc. of 50-percent Sorbitol intravenously, together with ice compresses locally and Demerol orally.

Within five hours, the pressure of the right eye rose to 72 mm. Hg and the left eye remained at 82 mm. Hg. By 10:30 p.m. the visual acuity was: O.D., 20/70; O.S., 20/50; and the intraocular pressure was: O.D., 53 mm. Hg; O.S., 23 mm. Hg. Subjectively the patient had improved. The pupils were still pinpoint and the eyes pale.

By four o'clock the next morning (April 19, 1948) the pain in each eye had recurred and at 7:30 a.m. the pressure was 82 mm. Hg in the right eye and 78 mm. Hg in the left eye. For the next four hours the pressure of the right eye varied from 63 to 73 mm. Hg and of the left eye 54 to 73 mm. Hg.

Since all attempted therapy had proven fruitless, surgical intervention was resorted to and, under general anesthesia, a bilateral Lundsgaard modification of the Holth iridencleisis was performed.

Following surgery, the anterior chambers remained extremely shallow, the pupils updrawn, and the eyes did not become soft until the third postoperative day.

On the sixth postoperative day the visual acuity was 20/70 in each eye and the intraocular pressure was 12 mm. Hg in the right eye and 15 mm. Hg in the left eye as measured with a Sklar-Schiøtz tonometer.

On the eighth postoperative day the patient was discharged from the hospital with the eyes finally moderately injected with a good filtering bleb present in each eye. The iris diaphragm was still up against the posterior cornea. The pupils were still drawn up. No synechias were visible. The only chamber visible was in the pupillary area. There was no evidence of a visible beam or cells in the anterior chamber of either eye. The iris pillars were well incarcerated and the ocular fundi were visualized and appeared normal.

The intraocular pressure on discharge measured 17 mm. Hg in the right eye and 14 mm. Hg in the left. The corrected visual acuity of the right eye was 20/50 and of the left eye 20/70.

Subsequent to discharge from the hospital, it took three weeks for the anterior chambers to reform, and the vision and pressure remained essentially the same as on discharge from the hospital. For the past four years the patient has been observed at frequent intervals.

When last examined on July 8, 1952, the corrected visual acuity of the right eye was 20/40 and of the left eye, 20/70. The intraocular pressure was 17 mm. Hg in each eye. The visual impairment was considered to be due to the updrawn pupils resulting from the surgical procedure. The eyes were pale. There was a moderate filtering bleb present in each eye. The anterior chambers were still very shallow. The chamber angle could not be visualized gonioscopically due to the high iris roll. The fundi could not be well visualized due to the updrawn pupils and the appearance of incipient lenticular opacities. The central and peripheral visual fields were within normal limits.

Comment

The use of six drops of 0.05-percent DFP in this patient whose anterior chambers were already shallow produced a rise in intraocular pressure in the right eye followed five hours later by a rise of similar proportions in the left eye. A thorough trial of medical therapy proved fruitless.

It was felt that an iridencleisis was indicated because the course up to the acute attack indicated increasing angle embarrassment which was not likely to respond to iridectomy alone. The mechanism seemed clearly to be that of a narrow-angle glaucoma. The subsequent course, with well-controlled pressure and a bilateral filtering bleb, proved the correctness of this supposition.

A remarkable feature of this case was the fact that, at the heighth of the attack, after

the use of DFP, the eyes were perfectly pale.

CASE 2

Mrs. E. B., aged 62 years, was seen by one of us (D. S.) on March 20, 1952, as an emergency case with acute glaucoma. She stated that she had had "iritis and keratitis" in 1912, and that since then she had been weak-sighted in spite of the use of corrective lenses.

She first noticed haloes in the left eye in October, 1951, but, since these were not particularly disturbing, did not seek attention until February, 1952. At that time she had her eyes tested but the "test was not satisfactory." She did nothing further until March 14, 1952, when she saw an ophthalmologist who found her intraocular pressure in the low 30's in each eye. He prescribed pilocarpine to be used as drops.

Because this did not seem to affect the intraocular pressure after one week's use, he changed to 0.1-percent DFP in peanut oil. This was prescribed on March 19, 1952, and the patient instilled two drops in each eye, one at about 5 p.m. and one at about 10 p.m. just before retiring. She was awakened at about 1 a.m. by severe pain in both eyes which persisted until noon of the following day when she was first seen. Concomitant with the pain there was marked diminution in vision.

At the time of the first examination the following findings were noted:

Vision was: R.E., 20/300, correctible to 20/200; L.E., finger counting at one foot, correctible to 20/300.

The correction she was wearing was:

R.E., −8.0D. sph. ⌒ −2.0D. cyl. ax. 45°
L.E., −8.0D. sph. ⌒ −2.0D. cyl. ax. 135°
Add +2.5D. sph., O.U.

Both eyes presented the same picture. There was bilateral slight mixed injection. The corneas were diffusely hazy as the result of epithelial edema so that details of the corneal structure and of those structures lying posterior to it could not be distinguished. There seemed to be no posterior corneal precipitates. The anterior chambers were moderately deep. The pupils were three mm., fixed and slightly eccentric.

Ophthalmoscopically a red reflex only was apparent, the left slightly better than the right. The intraocular pressure with the Schiøtz tonometer was over 80 mm. Hg in each eye.

In view of the experience with the previous case and because it was felt that medical therapy would not help, it was decided to do an immediate bilateral iridencleisis. This was done on March 20, 1952, and was technically uneventful, one pillar being incarcerated in each eye.

It was noteworthy that the retrobulbar injection just prior to surgery, consisting of two-percent procaine, 1:50,000 adrenalin, and 7.5 turbidity reducing units of hyaluronidase per one cc., considerably reduced the intraocular pressure.

The postoperative course was uneventful. The corneas cleared immediately. There was moderate postoperative conjunctival chemosis which subsided gradually.

At the time of discharge from the hospital on the eighth postoperative day, the conjunctival flaps were bilaterally boggy, indicating good filtration. The anterior chambers were normal in depth and optically empty. There were definite bilateral pigment synechias to the anterior lens capsule. The fundi were easily seen and the optic nerves showed no excavation. There was a small myopic crescent in each eye, and the retinal vessels appeared normal. Corrected vision was: R.E., 20/40; L.E., 20/100.

The patient was last seen on June 13, 1952. Her eyes were comfortable and her corrected vision was 20/50+ in each eye and five-point print. Both eyes were pale. At the 11-o'clock position in the right eye, there was a definite filtering bleb.

The cornea was clear. The anterior chamber was deep. The pupil was distorted by the coloboma. At the 12-o'clock position, there were some fine pigment deposits on the anterior lens surface. The lens itself showed some nuclear sclerosis but no "Glaukom flecken."

The fundus was easily seen and the disc was of good color, with no excavation. There was a small temporal conus but no other myopic changes.

The left eye presented a similar filtering scar. The cornea had some diffuse old stromal scars with several ghost vessels extending into the cornea from the 4-o'clock position. There were numerous fine pigmented deposits on the posterior cornea. The anterior chamber was optically empty and deep. There was a surgical coloboma at the 12-o'clock position with incarceration of the temporal pillar in the filtering scar. There were pigment deposits on the anterior lens surface. The lens showed somewhat more nuclear sclerosis than the right.

The left fundus was seen with somewhat more difficulty than the right, but seemed to be the same as the right.

The intraocular pressure was 17 mm. Hg (Schiøtz) in each eye. Gonioscopy was the same in both eyes and showed the angle to be narrow throughout with the first part of the trabeculum visible only in the lower portion. The trabeculum was not visible over the iris roll nasally and temporally. The visual fields showed slight constriction of the left visual field and slight enlargement of both blind spots.

COMMENT

When the patient was first seen and before gonioscopy was possible, the chambers seemed deep and it was felt that, unlike the first case, there was a wide-angle glaucoma present. At the earliest opportunity that gonioscopy was practical, it was found that this was one of the rather rare cases in which the chamber was of normal depth with a

narrow angle. The mechanism seemed clearly to be that of a narrow-angle glaucoma.

Iridencleisis was chosen because it was thought at the time that the patient had a wide-angle glaucoma and, further, that the glaucoma was sufficiently advanced prior to the onset of the acute attacks so that there was some question as to whether it could be controlled medically.

Discussion

These two cases re-emphasize a point which was brought out numerous times previously, namely, that DFP is capable of increasing the capillary permeability and at the same time of causing such a spasm of the ciliary body that the whole iris lens diaphragm may be shifted forward. It is plain that these factors may further embarrass an already inadequate chamber angle. These changes may so aggravate a narrow-angle glaucoma, as in the cases reported here and that reported by Butler, that the condition becomes medically irreversible and requires surgery.

McDonald, as previously mentioned, stated that DFP might hasten the need for surgical intervention, and we emphatically agree with this statement. Moreover, it is likely that surgery so forced is more difficult than it otherwise would be, since the intraocular pressure is higher and the anterior chamber more shallow than it would usually be.

It is unfortunate that so few of the reports distinguished between wide-angle and narrow-angle glaucoma, since this would appear to be an important consideration. Leopold and McDonald, who specifically advocate the use of DFP in narrow-angle glaucoma; Haas, who reports one case of narrow-angle glaucoma in which the pressure was increased by its use; and Butler, who reports its use in a narrow-angle glaucoma in a case quite similar to the two reported here, are the only authors who specifically mention the condition of the chamber angle.

McDonald stressed the fact that the patient should be kept under direct supervision for one hour after the first instillation of DFP in order to make sure that there is no immediate increase in intraocular pressure. This is a precaution that should be emphasized, although in our Case 1 it is unlikely that a pressure reading one hour after instillation of DFP would have revealed any increase in intraocular pressure, since the acute attack of pain did not have its onset until the fourth day after the drug was started.

After reviewing the literature and in view of the two cases reported here, it must be concluded that DFP is a drug which should be used with great caution. It is our feeling that it should not be used in narrow-angle glaucoma under any circumstance. It should be used in wide-angle glaucoma only after gonioscopy proves that the angle really is wide, and then only when the precaution cited by McDonald is followed:

Keep the patient under direct supervision for at least one hour after the instillation of the first drop to make sure there is no immediate rise in pressure.

The authors feel that the only real indication for the use of DFP is in aphakic glaucoma where, because of altered anatomy of the eye, the chamber-crowding mechanism is not important.

Summary

1. Two cases of narrow-angle glaucoma are reported in which a severe acute attack was produced in both eyes by use of DFP.

2. A review of the literature indicates that a paradoxical increase in intraocular pressure following the use of DEP is not uncommon in both wide- and narrow-angle glaucoma.

3. We believe DFP to be contraindicated in narrow-angle glaucoma.

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OPHTHALMOLOGY IN KOREA

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Evacuation

The Korean conflict has produced many new methods of approaching the problem of battle casualties, their treatment, and their evacuation. I am going to describe the organization of the ophthalmologic department of a medical battalion of a marine division, Fleet Marine Forces, and how ocular casualties and diseases are being handled.

The chain of evacuation is the most important factor in bringing the wounded marine to an area where he can receive definitive therapy. A hypothetical case of a casualty with a corneal laceration will be presented.

A small patrol reports back to the company headquarters. The corpsman reports that one of the men has been hit in the right eye by a fragment from an enemy grenade. The man is on a stretcher with both eyes patched. The corpsman has attached a tag to the wounded man's clothing stating the time and nature of the wound. If the man were in pain, the morphine dosage and the time of its administration would also be given. The company commander then phones the aid station and reports the casualty.

An ambulance brings the patient to the aid station where he is examined by a doctor, who gives further first-aid treatment. This case is a corneal laceration. He again patches both eyes and calls for a helicopter. The helicopter arrives and the patient is flown to the division hospital where he is placed on the operating table after diagnostic studies and examination.

The total elapsed time from the first reporting of the wound to the arrival of the

casualty at the division hospital usually averages 30 minutes. This amazing rapidity in evacuation is saving many eyes in Korea.

The most prominent factor in rapid evacuation is the helicopter. Too much praise cannot be given the helicopter and its pilots. The jostling of ocular patients in an ambulance on the rough Korean roads would no doubt be disastrous to vision in many cases.

The facilities at the field hospital may often be inadequate to treat casualties. In this event, the patient is sent from the hospital, via helicopter, to a naval hospital ship, which is well equipped to handle the cases. The hospital ship also handles the special postoperative nursing care that is required by casualties with ocular injuries. The surgeon from the field hospital can make postoperative visits to those patients on whom he has operated at the field hospital.

The division hospital

The first definitive ocular establishment in the chain of evacuation is at the division hospital, where the division ophthalmologist is located. The eye clinic at this unit has most of the facilities to be found at any well-equipped clinic, even though it may be located near the front lines.

The marine division eye clinic consists of a large room in a one-story metal-covered building. The refracting range is located on one side of the room partitioned off to make a four-foot width and is 20 feet long. The power for the AO projector and for the lights comes from a field generator.

Patients who break their glasses and have a visual acuity of less than 20/20 to 20/25 may be refracted and will receive glasses within seven days from an optical unit located to the rear. Two pairs will be issued in most cases.

For the most part, manifest type of refraction is done unless there are definite indications for cycloplegia. The range includes the screen for the projector plus a Lancaster-Regan dial set, illuminated by 20 foot-candles of light. The retinoscope used is a Copeland streak retinoscope.

An individual whose naked visual acuity is less than 20/50 receives two pairs of glasses. The frames furnished are zylonite plastic or gold-filled metal, depending on the patient's preference. Those patients on whom a cycloplegic has been used are not sent back to a line unit, if that is their station, for obvious reasons.

Patients are given physical profiles according to standards set up by the Department of Defense which denotes their physical status. The visual requirements are such that no man is assigned to duties which are not compatable with visual acuity. Marines are all screened before being sent overseas and glasses are issued to them at that time when necessary.

Elective surgical procedures, for the most part, are not being done in Korea. Pterygiums, strabismus, and lid plastic surgery are in this category. We have curetted many chalazia, which seem to be relatively common among the troops. A pterygium will be operated if it has encroached on the cornea four to five millimeters or if it is causing obvious irritation.

Intraocular foreign bodies

The most prominent casualty is due to an intraocular foreign body, usually a metallic fragment from an explosion of mortars or grenades. Those foreign bodies which can be removed without too much intraocular trauma are removed at the division hospital.

The first step in treatment is localization of the foreign body. This is usually done with a lateral and a stereo-Waters view by roentgenogram. The stereoview is observed with an acrylic-resin prism coated on its base. This enables one to have a stereoptic view of the foreign body and its orbital location. Of further aid is the injection of air into the space under Tenon's capsule. We also have the equipment for Sweet X-ray localization.

If the foreign body is deep in the eye, the patient is transferred to the hospital ship for removal of the fragment. A cauterization unit

can be set up in the field but the danger of infection in extensive intraocular procedures is high in Korea and the hospital ship is preferable.

Often the fragments lie outside of the globe in the orbital cavity. When they do, they are removed directly with the use of a Lancaster hand-magnet.

A great many eyes are so damaged that the only surgical procedure is enucleation. In these cases, a very extensive study is made before operative procedures. The Berens' glass sphere is inserted and Tenon's capsule sutured over it, followed by closure of the conjunctiva. These patients are sent to the ship for the postoperative care and then are evacuated to Japan. It should be emphasized that most of these patients have multiple wounds and while surgery is being performed on the eye there is other surgery elsewhere on the body.

The division ophthalmologist is equipped with a mobile kit containing all the required surgical equipment for eye surgery and he can be called to any of the division hospital units on the marine front.

Types of injuries

Corneal lacerations are usually sutured with 6-0, black-silk suture and covered with a conjunctival flap. According to the location of the laceration, the sliding flap is often used. Iris prolapses are excised when they occur.

Lid lacerations are common casualties. These are repaired with 6-0 silk suture as soon as possible. Patients with extensive lacerations of the face and lids that require grafting are flown to the hospital ship. Traumatic cataracts are quite common when the wounds involve the anterior segment of the eye. Cortical material is irrigated via a limbus-based incision.

One of the most prevalent injuries in Korea has been flash burns of the eye. These are caused by gasoline heaters for the most part. They usually result in a small staining area of the cornea and mild conjunctival injection which are treated with atropine, tetracaine, and patching, and clear up in about 48 to 72 hours.

Concussion cases are often accompanied by the signs of contusion to the eyeball. These are prevalent in blast injuries. Hyphema is common and is treated with bedrest and bilateral patching. Blast injuries may involve the iris, lens, retina, choroid, conjunctiva, cornea, and lids with various injuries.

Involvements of the iris, particularly iridodialysis, have been the most often seen. Lid ecchymoses and chemosis often accompany these injuries. Strangely enough, we have seen no dislocations of the lens or ruptures of the sclera due to blast.

Commotio retinae has been prevalent. Many of these cases have had a dilated, fixed pupil as the only resulting complication. The intraocular pressure has never risen above 33 mm. Hg (Schiøtz) in those cases we have seen and usually has returned to normal in 48 hours.

Conjunctivitis

Conjunctivitis is prevalent after the onset of cold weather. The cases are mild, for the most part, with staphylococci being seen on smear. These mild cases have usually been accompanied by blepharitis. They have responded well to treatment with sodium sulfadiazine in 10-percent solution in addition to local heat.

A more severe case of conjunctivitis often produces marginal infiltrations of the cornea which do not stain with fluorescein. These respond well to a combination of systemic and local aureomycin in addition to local heat. In the more resistant cases, topical cortisone, when available, has sometimes produced remarkable results.

No specific organism has been cultured or seen on smear in these cases of keratoconjunctivitis. We have a universal-type slit-lamp available for examination of the anterior segment of the eye, valuable in diagnostic studies.

Irrigation of lacrimal sacs with the 10-percent sulfadiazine solution has produced

good results in those cases of chronic conjunctivitis with repeated exacerbations. These patients return to the eye clinic two to three times a week for dilation and irrigation.

Foreign bodies are removed from the cornea with a spud under local pontocaine anesthesia. Many fragments cannot be removed readily with any instrument, including the hand-magnet. We have left many foreign bodies, which have become covered with epithelium, with no untoward effects on vision recorded to date. Often extensive opacified sites have cleared under topical cortisone.

The drugs available are atropine, aureomycin, sodium sulfacetimide, eserine, boric acid, sulfathiazole, ammoniated mercury, all in ointment form. In solution, in the clinic, there are aqueous zephiran, homatropine, atropine, pilocarpine, eserine, sodium sulfadiazine, all in droppered bottles. A host of other drugs are available at the division pharmacy, located near by and prescriptions are readily filled.

No cases of glaucoma that were not secondary to injury have been noted. All battalion surgeons are well informed as to the signs of glaucoma.

Angioneurotic edema has been the most prevalent allergic ocular condition. This has increased with the change over to woolen clothing and fur-lined hoods. These cases have all responded well to antihistamine drugs.

One danger prevalent in Korea has been methyl-alcohol poisoning. Fortunately, there have been very few cases and we have seen no blindness resulting from such intoxication in the marine division. We saw 12 cases of methyl-alcohol poisoning in South Korean Service Corps personnel. There were many fatalities in this group. The one patient retained at the division hospital became blind. The blindness lasted for five days, after which the patient developed tubular vision which cleared in two days. There was full recovery.

South Korean forces

The Korean forces serving with the marine division have suffered from no diseases, from an ocular viewpoint, that would call for special attention. One finding, which is not unusual in oriental races, is axial myopia. It was present in a large number of the South Koreans.

The South Korean Marine Corps personnel are refracted in our clinic and the prescription is filled by the Korean naval medical unit. A great many of the surgical patients are South Korean and they are often sent on to the hospital ship, according to the extent of the wounds.

Any equipment needed by the eye department can always be obtained without much delay. The facilities available for ophthalmologic care of patients are most adequate.

Summary

A report is made on the ophthalmologic facilities available in treating patients of the First Marine Division in Korea. One of the most advantageous things is the speedy, and nontraumatic evacuation of eye casualties by helicopter. The close liaison between the field hospital and the hospital ship enables casualties to receive excellent care.

10 Grand Avenue.

SPASMUS FIXUS WITH CEREBRAL PALSY*

A CASE REPORT

GEORGE GUIBOR, M.D.
Chicago, Illinois

I can find no case reports in which spasmus fixus and cerebral palsy have been associated, and in which ocular abduction ability has improved following the use of atropine cycloplegia and prism glasses. Therefore, this case report warrants our consideration.

CASE REPORT

This girl, a first child, was born prematurely on August 8, 1948, two and one-half months before term. She weighed two pounds and two ounces at birth. Delivery was normal. She was in an incubator at Michael Reese Hospital for 80 days. The right eye deviated toward her nose and this condition became more severe following an attack of pneumonia when she was four months of age.

When examined in the eye clinic at Children's Memorial Hospital, August 8, 1949, both eyes deviated toward the nose and neither eye could be abducted (Fig. 1—A, B, and C). A diagnosis of bilateral abducens palsy was suggested. She was referred to the clinic for oculomotor anomalies, October 17, 1949.

Atropine retinoscopy was: R.E., +2.5D. sph. ⌒ +1.5D. cyl. ax. 90°; L.E., +2.75D. sph. ⌒ +1.75D. cyl. ax. 90°. She was given the following prescription: R.E., +1.25D. sph. ⌒ +1.5D. cyl. ax. 90° with 5Δ base-out; L.E., +1.5D. sph. ⌒ +1.75D. cyl. ax. 90° with 3Δ base-out.

Gradually, she developed a slight movement of each eye laterally after atropine cycloplegia, but both eyes deviated nasally 35 to 40 degrees most of the time, so that instead of moving her eyes laterally she moved her head from side to side. When either eye was occluded, a monocular horizontal nystagmus occurred so that I interpreted her vision to be less than 20/200 (20 percent) in each eye.

The diagnosis at this time (October 17, 1949) was: (1) Spasmus fixus, and (2) spastic esotropia.

Atropine was placed in each eye four nights each week from April 24, 1950, to December 4, 1950 (eight months).

Frequent examinations were done to ascertain the possibility of her developing abduction ability in each eye. At times this ability to gaze laterally was almost normal. At other times it was approximately only 50 percent of the normal (Fig. 1—D, E, F, and G).

The patient by this time was 28 months of age. She began to sit up only when 15 months of age. She tried to walk at 20 months but could not do so. She was, therefore, referred to the Orthopedic Clinic and Neurologic Clinic, with a diagnosis of cerebral palsy, hypotonic type.

Both clinics reported that all deep reflexes were depressed and slight shortening of the Achilles tendon existed. The muscles of her ankles and feet were hypotonic. The Babinski extension reflex was at first absent.

The diagnosis was: (1) Double hemiparesis with hypotonia, (2) spasmus fixus, and (3) spastic esotropia.

She began to talk February 11, 1951, when she was two and one-half years old. She could move her eyes satisfactorily but was not walking. To improve ocular movements and to decrease convergence spasms, atropine was used in each eye every night for three additional months. The ocular movements improved, but subnormal searching fixation persisted in the right eye.

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* From the Department of Ophthalmology and Neurology of Children's Memorial Hospital. Presented before the Chicago Ophthalmological Society, January, 1953.

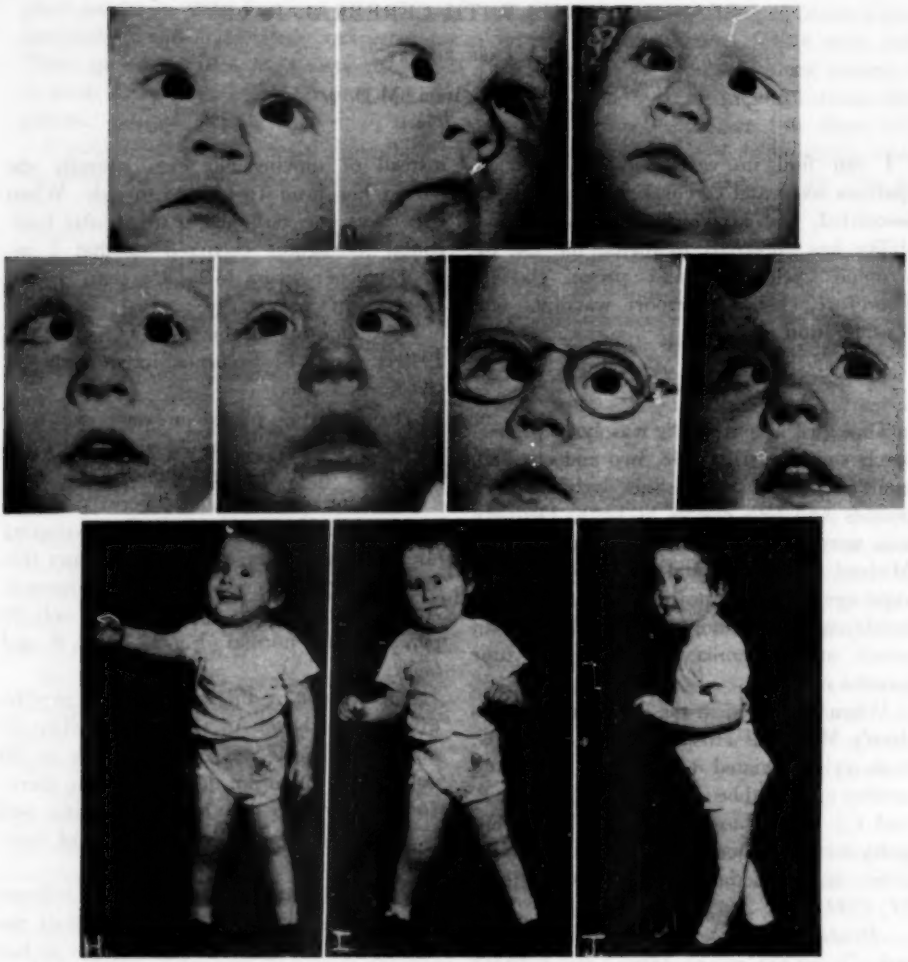

Fig. 1 (Guibor). Bilateral fixed convergent strabismus in a four-month-old infant with cerebral palsy.
(A) Spasmus fixus. Note both eyes converging simultaneously.
(B) Inability to gaze to right with right eye.
(C) Inability to gaze to left with left eye.
(D) Ability to gaze to left with left eye, developing after use of atropine.
(E) Ability to gaze to right with right eye, developing after use of atropine
(F and G) Abduction, left eye, almost normal.
(H) Appearance of stance, with wide base and overextension of knees.
(I) Attempting to walk, with increased wide base and overextension of knees.
(J) Side view. Note overextension of left leg as a symptom of hypotonic type of cerebral palsy.

An interesting improvement appeared in her walking January 9, 1952. Dr. Buchanan, the neurologist, noticed at this time that deep reflexes in her arms and legs were appearing. Her Babinski reaction was present in extension, and she walked with a wide base and very stiffly (Fig. 1—I). The Orthopedic Department prescribed stretching exercises for her legs, so that the contractures of her Achilles tendon gradually decreased and later could not be found (February 11, 1952).

Present status. She can now talk well, walks stiffly with a wide base and overextended knees (Fig. 1—H, I, and J). She has normal comprehension. Her visual acuity has developed until it is now 20/20. She still has a spastic variable esotropia (Fig. 1—H).

Discussion

This patient possessed strabismus, spasms of both internal rectus muscles, and weakness of both external rectus muscles before a definite diagnosis of cerebral palsy was made. Abduction ability of each eye developed and at times is normal.

The history of prematurity, slow motor development, and abnormal eye motor coordination suggested to the Ophthalmologic Department of Children's Memorial Hospital that this patient had a double hypotonic hemiparesis. The diagnosis was confirmed by the Orthopedic and Neurologic Departments.

It is interesting to note that eye motor defects exist in over 50 percent of patients who have cerebral palsy. Is it possible that the central nervous system could be responsible for these cases with oculomotor defects?

30 North Michigan Avenue (2).

RETINOBLASTOMA TREATED WITH INTRAVENOUS NITROGEN MUSTARD*

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Baltimore, Maryland

Intravenous nitrogen-mustard therapy has been successfully employed in the treatment of the leukemias, lymphomas, and neuroblastomas. It has apparently not been used heretofore in the treatment of retinoblastoma. Its use in this case was suggested by Dr. Francis Schwentker, professor of pediatrics in The Johns Hopkins University. The treatment was undertaken after consultation between Dr. Schwentker and Dr. A. C. Woods.

Report of case

The patient is a six-year-old white boy whose right eye had been enucleated at the age of 15 months for what was clinically diagnosed as a retinoblastoma. Microscopic sections showed a typical retinoblastoma (fig. 1). The left eye was examined periodically and remained entirely normal with a vision of 20/20, the last examination being made when the patient was five and one-half years of age.

Routine school examination on the patient's sixth birthday revealed that the vision in the left eye had fallen to 20/50. Two weeks later, the vision in the left eye had dropped to 20/200 and there was marked field loss. The patient was referred to the Wilmer Institute and seen for the first time December 3, 1952.

On admission examination, there was anophthalmos on the right with a good-fitting prosthesis and a clean socket. Vision in the left eye was 5/200. The pupil was widely dilated (mydriasis) and, with the hand light, there was an obvious detachment below, nasally and temporally. Tactile tension was normal.

* From the Wilmer Ophthalmological Institute of The Johns Hopkins Hospital and University.

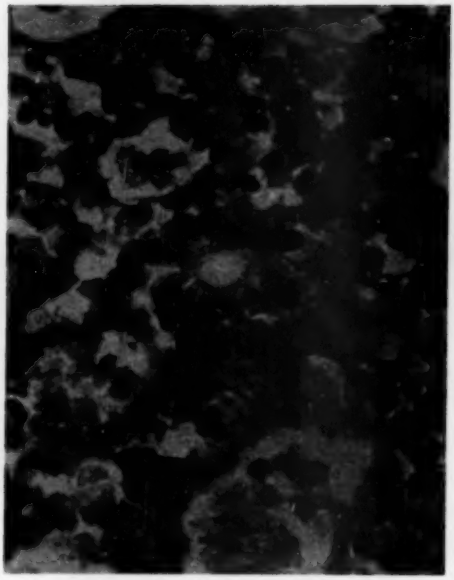

Fig. 1 (Kupfer). Photomicrograph of section of retinoblastoma from the right eye which was enucleated when the patient was 15 months of age (×600).

On slitlamp examination, the anterior segment was normal. There were several vitreous floaters suggestive of cellular debris and a vitreous detachment was noted.

On ophthalmoscopic examination, the entire retina, with the exception of a small strip in the upper nasal quadrant, was elevated in a big billowing detachment. The disc appeared blurred in outline and was buried in the depths of the billowing folds. The veins were markedly distended and tortuous. The arteries were normal.

Fig. 2 (Kupfer). Fundus drawing before therapy.

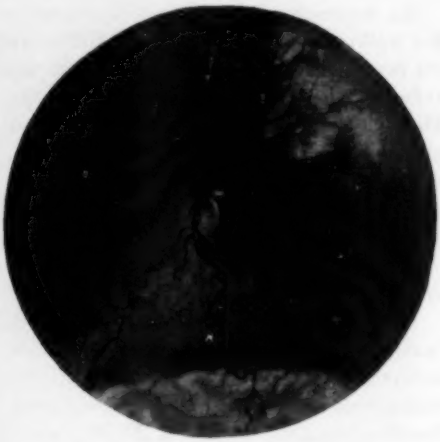

Fig. 3 (Kupfer). Fundus drawing 14 days after therapy.

The detached retina was freely movable except temporal to the nervehead where there was a large diffuse whitish mass. This mass merged with the detached retina temporally.

Inferiorly, the surface of the retina appeared cystic. Superiorly, along the strip of attached retina were several large plaques of chalky white material (fig. 2).

There was no X-ray evidence of bony metastases. Soft tissue films of the globe revealed no calcium in the tumor. It was decided to combine the usual radiation therapy with intravenous nitrogen mustard.

Concomitantly with irradiation of the tumor according to the method of Reese, a course of intravenous nitrogen mustard was begun. Four-tenths of a milligram per kilogram body weight of nitrogen mustard was given in divided doses over an eight-day period for a total dose of 9.5 mg.

Within five days after the therapy had been started, the fundus picture began to im-

prove and by the 14th day the billowing detachment had entirely disappeared, leaving the retina moderately elevated below, where it appeared more pigmented and cystic. Temporally, the tumor mass was well delineated in the region of reattached retina and a small area of superficial hemorrhage lay over the tumor mass (fig. 3).

Fifty days after commencement of therapy, the retina was completely reattached. Diffuse pigmentary changes (especially in the lower temporal quadrant) and evidence of calcium were noted, both being characteristic of regressed retinoblastoma* (fig. 4).

The irradiation was discontinued at this point because of the dramatic response to the nitrogen-mustard treatment and to avoid the increasing danger of late vitreous hemorrhage and other complications at higher radiation dosage levels. A total skin exposure of 4,450 r × 2 (calculated tumor dose 4,850 r) had been given over a period of 50 days. The vision in the left eye at this time was 20/200.

Three weeks after the completion of the X-ray therapy, the patient was readmitted to the Wilmer Institute and received a second and final course of intravenous nitrogen mustard, 9.3 mg. being given in divided doses over a four-day period. The vision in the left eye at time of discharge February 16, 1953, was 20/100, the visual field 40 degrees.

Fig. 4 (Kupfer). Fundus drawing 50 days after therapy.

* Personal communication from Dr. Algernon B. Reese: Dr. Reese saw the patient at this time and stated ". . . this is a completely regressed case. . . . I have never seen such a remarkable regression as this and certainly this is more than could be expected from X ray alone."

Addendum

On October 16, 1953, the patient's vision was reported to be 20/30-2, and there was no evidence of recurrence of the tumor.

The Johns Hopkins Hospital (5).

Ophthalmic Miniature

If a person has cataracts in his eyes and one of them is shut, the hole in the other is enlarged; and this is proof that the power of vision has been left normal. If anyone's pupil is not enlarged when one eye is shut, then the power of vision is destroyed at the source; for this reason it is vain that you hope to perform a successful couching operation on his eye, for he will not see.

Hunain Ibn Is.-Haq. (809-877),
The Book of the Ten Treatises on the Eye.

AN EVALUATION OF CYCLOGYL (COMPOUND 75 GT)*

BENJAMIN MILDER, M.D., AND RALPH S. RIFFENBURGH, M.D.
Saint Louis, Missouri

Reports of a new cycloplegic drug, Compound 75 GT (Cyclogyl†) have indicated that it is superior to homatropine, having a more rapid onset, shorter duration, and more intense cycloplegic effect.[1] Stolzar[2] found an average residual accommodation of 1.03 diopters one hour after using this drug. Gettes and Leopold[3] found, for Negroes, that Cyclogyl produced a residual accommodation of 2.5 diopters or less in two thirds of their patients.

Cyclogyl is a spasmolytic agent of the class of basic esters of substituted phenyl acetic acids. This study employed a 0.5-percent solution in a boric acid-borate buffer of pH 5.7 with 1:50,000 Zephiran added as a preservative.

A study of Cyclogyl (0.5 percent) and homatropine (five percent) was undertaken in order to compare the following characteristics of the two drugs.

1. Relative rapidity of onset of maximum cycloplegia
2. The duration of maximum cycloplegia
3. The rapidity of dissipation of the cycloplegic effect
4. The mydriatic effect

Fifty routine refraction patients were selected from both clinic and office practice, ranging from eight to 48 years of age. Twelve of the patients were Negroes.

Cyclogyl was instilled into one eye of each patient twice, 10 minutes apart. Homatropine was similarly instilled twice into the other eye. In the eye which received the homatropine, one drop of one-percent paredrine was also instilled. Taking the time of the first instillation as the zero time, the pupillary size and the amplitude of accommodation were measured for each eye at 15-minute intervals for the first hour; then at one and one-half, two, and three hours.

While most patients complained more of stinging on instillation of Cyclogyl than of homatropine, in no case was there hyperemia of the eye receiving Cyclogyl. About one fourth of the patients showed definite conjunctival hyperemia of the homatropine-treated eye.

MYDRIATIC EFFECT

Onset of pupillary dilatation was noted sooner with Cyclogyl in almost all cases. The maximum pupillary size with Cyclogyl was reached at an average time of 41 minutes. With homatropine-paredrine, the average time was 51 minutes.

The maximum size reached with homatropine-paredrine was usually larger, averaging 7.4 mm., while the Cyclogyl-treated pupils averaged 6.7 mm.

* From the Department of Ophthalmology, Washington University School of Medicine, and the Oscar Johnson Institute. Presented before the Saint Louis Ophthalmological Society, January, 1953.

† Supplied through the courtesy of E. W. Blanchard, Ph.D., Director of Research, Schieffelin and Company, New York.

TABLE 1

ANALYSIS OF CYCLOGYL

	Average Minimum Residual Accommodation		Time of Onset (minutes)		Duration (minutes)	
	Homatropine	Cyclogyl	Homatropine	Cyclogyl	Homatropine	Cyclogyl
Negroes	3.1 D	2.4 D	60	50	72	33.5
White	1.4	1.3	72	46	70	34.5
Total	1.83	1.58	69	47	71	34

Although recovery begins sooner with Cyclogyl, as indicated by the fact that many of the pupils had begun to contract by the end of three hours, complete recovery to normal was prolonged and many eyes still showed some mydriatic effect the morning after refraction. None of the homatropine-paredrine treated pupils had begun contraction within the three-hour observation period.

In Negro patients, the maximum size of the pupil was smaller and the time required to reach the maximum effect longer, with both drugs.

Cycloplegic effect

The accommodation was measured with the Prince rule to the nearest one tenth of a diopter, with the best accuracy obtainable. Minimum accommodation was considered to persist as long as there was less than 0.25 diopters increase from this minimum value. Table 1 shows that, while the minimum accommodation with Cyclogyl averaged 0.25 diopters less than with homatropine, the difference in white patients was not significant. In Negro patients, neither drug produced consistent adequate cycloplegia.

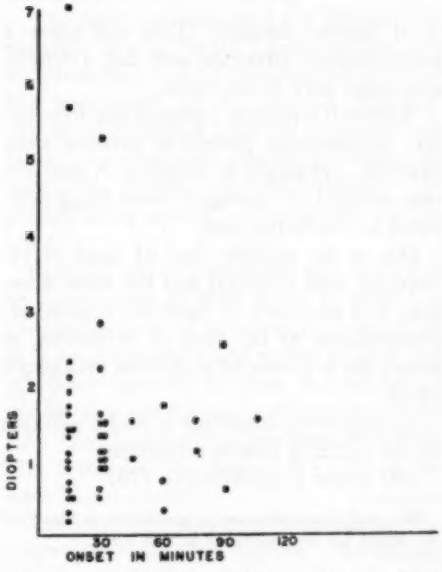

Fig. 1 (Milder and Riffenburgh). *Cyclogyl.* Time of onset of maximum cycloplegic effect and amount of residual accommodation at this time, plotted for each Cyclogyl-treated eye. The circles represent white patients, the black squares, colored, in each figure.

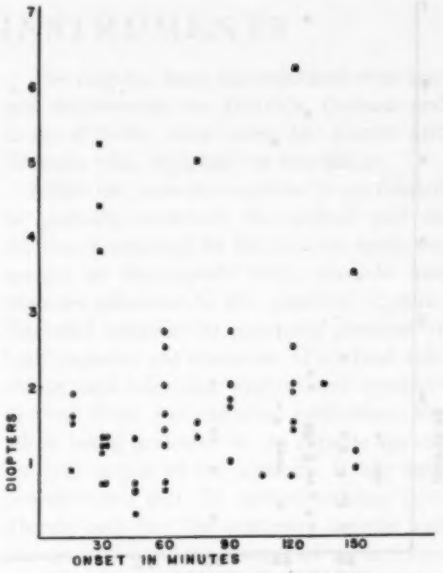

Fig. 2 (Milder and Riffenburgh). *Homatropine.* Time of onset of maximum cycloplegic effect and amount of residual accommodation at this time, plotted for each homatropine-treated eye.

Table 1 further demonstrates the significantly more rapid onset of maximum cycloplegic effect and its shorter duration, with Cyclogyl. If the cases are plotted individually on a scatter graph (figs. 1 and 2), it is shown that 40 percent of the Cyclogyl-treated eyes reached maximum cycloplegia in 15 minutes or less, while 22 percent did not attain the maximum effect until 45 minutes or more.

Similarly plotting the duration of maximum cycloplegia (fig. 3), it can be seen that 30 percent of the Cyclogyl-treated eyes have a maximum cycloplegic effect lasting 30 minutes or less. Thus, in a significant number of the eyes treated with Cyclogyl, the optimum time for refraction was 45 minutes or less; on the other hand, 22 percent of these eyes did not reach their maximum

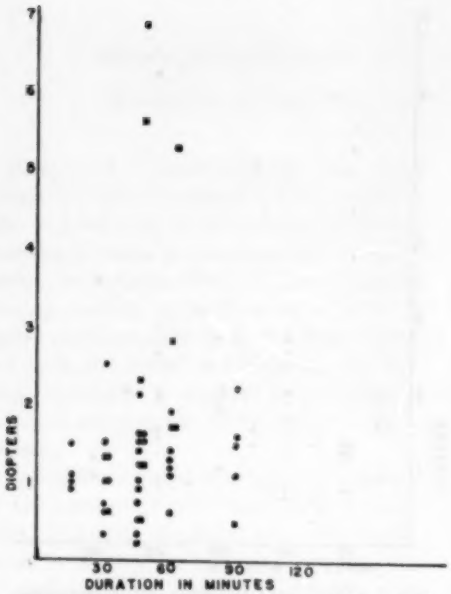

Fig. 3 (Milder and Riffenburgh). *Cyclogyl.* The duration of maximum cycloplegia and its amount, plotted for each Cyclogyl-treated eye.

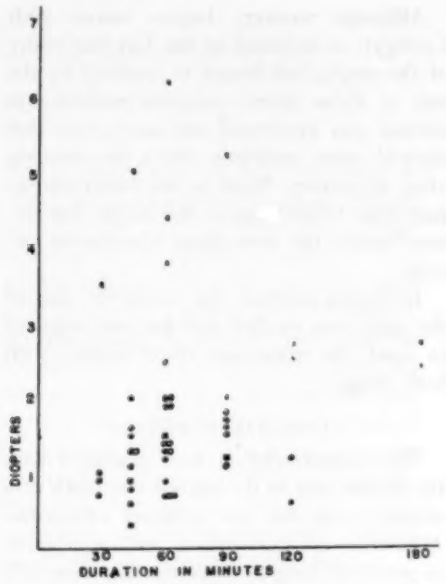

Fig. 4 (Milder and Riffenburgh). *Homatropine.* The duration of maximum cycloplegia and its amount, plotted for each homatropine-treated eye.

effect until at least 45 minutes had passed.

Because of this variability, we feel that it is important to measure the residual accommodation routinely, at the time of refraction. Because of the longer duration of cycloplegia with homatropine (fig. 4), routine checking of the residual accommodation is of less practical importance.

The cycloplegic effect of Cyclogyl had disappeared in 24 hours, while many homatropine-treated eyes retained some effect. Many patients stated that they could read on the evening of the refraction with the Cyclogyl-treated eye but that the other eye remained blurred.

Conclusions

Cyclogyl (0.5 percent) compares favorably with homatropine in cycloplegic effect for refraction, produces the effect sooner, and is of shorter duration. This will allow a postcycloplegic effect the next day, a definite advantage over homatropine.

Neither 0.5-percent Cyclogyl nor five-percent homatropine proved to produce truly effective cycloplegia in Negroes. A one-percent solution of Cyclogyl is now being evaluated for use in this race.

Due to the variable time of onset of cycloplegia with Cyclogyl and the short duration, it is necessary to check the residual accommodation at the time of refraction, to insure the presence of maximum cycloplegic effect.

Cyclogyl will constitute a useful addition to our available aids to refraction.

640 South Kingshighway (10).

We wish to express our appreciation to Dr. Paul W. Miles for his assistance in this study.

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NOTES, CASES, INSTRUMENTS

SPONTANEOUS FORWARD DISLOCATION OF SOEMMERING'S RING*

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Mrs. A. M., aged 58 years, attended the out-patient eye department at the Leeds General Infirmary on November 6, 1951. The patient attended for refraction only and was not concerned for her right eye which had been blind since the age of 17 years. The history was that in 1948 she was found to have a traumatic cataract from a dart injury in 1911 (right eye).

When examined on November 6, 1951, vision was: R.E., no light perception; L.E., with a +0.25D. cyl. ax. 180° = 6/18 plus; add +3.0 = J4.

Tension was: R.E., 10 mm. Hg (Schiøtz); L.E., 28 mm. Hg.

The right eye presented the peculiar appearance seen in Figure 1 from the presence of a complete ring of Soemmering of calcified appearance in the anterior chamber. The dislocation had occurred spontaneously three years previously and the eye had never shown the slightest sign of irritation. The tension being normal, no treatment was advised.

Ringlike after-cataract was first described by Soemmering in 1828. It can be defined as a toruslike form of after-cataract consisting of cortical remnants and newly formed lens substance encysted between the residue of the anterior and the posterior capsules of the lens. The absence of nucleus is characteristic. The formation lies behind the iris, in the retro-iridial space.

This ring may develop after needling, Fukala operations for myopia, discissions, extracapsular extractions, or perforating wounds affecting the central part of the anterior capsule.

The ring has been demonstrated after animal experiments by Dietrich, Cocteau and Leroy-d'Etiôle, and later by Gonin and Wessely who explained its formation.

After the anterior capsule is perforated or partially removed, the central part of the lens is attacked by the aqueous while the margin of the capsule folds, retracts, and becomes adherent to the posterior capsule. Encysted between the equatorial portions of both capsules are remnants of cortical substance, and later also regenerated products derived from the capsular epithelium, the whole being protected by the capsule against the lytic action of the aqueous. It has been demonstrated that the epithelium may proliferate and line the posterior capsule and also grow around the margin of the anterior capsule. The cells resulting from this proliferation secrete a hyaline substance, the result being a number of hyaline layers separated by layers of cells.

Obviously the products of regeneration must play an important role in the formation of the ring, since it is unlikely that a mere encystment of coagulated lens remnants would form a ring of the dimensions of those which have been described.

The development of the ring has been observed after discission for secondary cataract; after the discission the capsule curls and retracts and with the release of internal

Fig. 1 (Arruga). Dislocated ring of Soemmering.

* From the Ophthalmic Department of The General Infirmary at Leeds.

tension proliferation may restart (Berliner).

It has been pointed out (Poos) that the younger the subject when the extraction or discission is performed, or the perforating wound occurs, the greater is the regenerative activity of the lens substance. This assertion coincides with Wessely's conclusions from his studies of experimental animals. He observed that the younger the animal where the extraction of the lens was practiced the greater the regenerative proliferation at the equatorial zone of the lens nucleus. The size of the ring depends on this activity.

The encysted matter may degenerate into calcareous and even osseous formations.

The ring is not always complete, being more often formed by several separated irregular masses.

There is a resemblance between the ring of Soemmering and the congenital form of disc-shaped cataract described, among others, by Vossius, Marchessani, and v. Szily; the latter author called it "lifebuoy" cataract and noted that the center of the ring was the same size as the missing nucleus. He observed this anomaly in four children of the same family. The cause would be either a complete aplasia of the nucleus or a secondary degeneration of the central area.

According to Ida Mann "the nucleus would have failed to develop and, shrunk and calcified, remained adherent to the capsule so that the outer fibers could not grow around it but merely apply themselves to the equatorial region."

According to Vossius the cause might be nutritional as a result of a central corneal perforation; the cells of the equator would continue to develop, forming normal fibers.

The ring of Soemmering is only seen clinically after dislocation, in cases of coloboma of the iris, where a total iridectomy has been performed, or occasionally with a very dilated pupil. In a very interesting case of Pavía and Dusseldorp where traumatic aniridia co-existed, the ring of Soemmering was visible in situ.

Usually the ring remains occupying the equator of the retro-iridial space, but in some rare cases it may dislocate either into the vitreous or the anterior chamber, or partly in both.

Cases of dislocation in the vitreous have been reported by Wessely[1] (1910) and Poos[5] (two cases, 1931).

Dislocation partly into anterior chamber, partly in vitreous, have been described by Pavía[6] (1931) and Tooke[7] (1933).

Adam[2] (1911), Schneider[3] (1927), Jess[4] (two cases, 1931), Nègre[8] (1933), Jacoby and Wolpaw[9] (1935), and Guha[10] (1938) published cases of anterior luxation of the ring (either complete or incomplete).

Dislocation may be spontaneous or determined by trauma. In the cases so far reported all dislocations into the anterior chamber were spontaneous (only in one of Jess's was there an antecedent of trauma seven months before the luxation). In all the others, with the single exception of Wessely's case, the luxation was caused by trauma.

According to Poos the dislocation would be facilitated by the fluidity of the vitreous, the zonular degeneration, and the weakness of the ciliary muscle, all characteristic of malignant myopia; and would also be related to the age of the patient when the lenticular trauma occurred: the younger the subject the greater the possibilities of development of a thick heavy ring and thereby of luxation.

Review of the cases so far published confirms that complete rings appear preferentially in patients in whom trauma occurred during infancy or youth and also in myopic eyes, both conditions being more commonly present where posterior dislocation took place. It is curious that in none of the cases of anterior luxation, some of which were moderately myopic (only one highly myopic) and the others nonmyopic, the dislocation was preceded by trauma;* while in most of

* In two cases only total iridectomy is quoted. It is possible that, though not mentioned, it had been practiced in one or two others.

the cases where malignant myopia was present (that is, those where the luxation was either partially or totally in the vitreous) traumatism was required to produce the dislocation of the ring. Also in this latter group the average time lapse between the lenticular trauma and the dislocation was greater.

Though no definite statistical conclusions can be derived from only 13 cases, these facts are in contradiction to the general belief, though the latter may appear more logical.

Posterior dislocation may be asymptomatic or cause visual disturbance. In one of Poos's cases vision was normally 6/12 but when the patient inclined the head the ring occupied the pupillary area and the eye was temporarily blinded. Anterior dislocation may, in addition, produce glaucoma. This was present in four of the eight cases of anterior luxation, doubtful in another one, and the remaining three had normal or low tension. Hypertension disappears with the extraction of the ring through a keratotomy (Jess). One case showed ciliary injection. In five cases the ring was removed either for hypertension, diminished vision, or ciliary injection, and in all improvement was obtained. In one of Jess's cases the eye had to be excised for absolute glaucoma.

I do not describe any further details of the cases so far appearing in the literature, since G. S. Guha in a recent paper (*British Journal of Ophthalmology*, April, 1951, pp. 226-231) has published a very complete table containing the most interesting data of their clinical histories.

I am indebted to Mr. John Foster for permission to publish the case of Mrs. A. M. who was seen in his clinic.

Pasaje Mendez Vigo 3.

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THE EQUIVALENT SPHERE AND CHROMATIC INTERVAL IN ASTIGMATISM

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The cross cylinder, whether used as a complete test or merely as an auxiliary check test, works best when the eye is in a condition of meridional balance. By this is meant that the astigmatism is changed into equally mixed astigmatism before the cross cylinder is applied.

The two focal lines of Sturm's conoid are then on opposite sides of the retina, which sort of straddles the conoid and has on it the interfocal circle (circle of least confusion). The position of the retina is "balanced" between the two principal meridians which have opposite and equal refractive status.

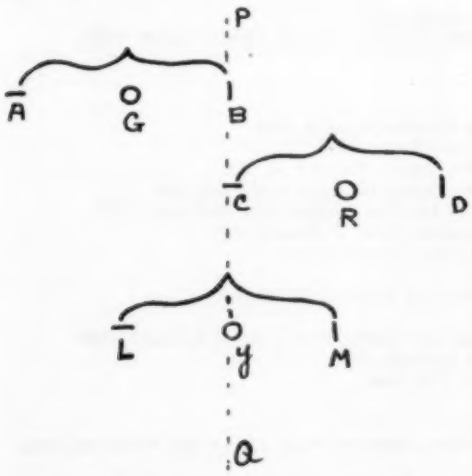

Fig. 1 (Pascal). Astigmatic interval, 1.0D. Chromatic interval, 1.0D.

Where the cross cylinder has to be used as a complete test, that is, where no preliminary test is available, the first step is to find whether or not there is any astigmatism. This is most easily found if the spherical lens already before the eye is the spherical equivalent or, better called, the equivalent sphere.

The equivalent sphere, it may be recalled, is the sphere which replaces a cylinder or spherocylinder and places the eye in meridional balance. It is equal to the algebraic sum of the sphere and half the cylinder. Thus if an eye requires, say, a +1.0D. sph. ⌒ +2.0D. cyl. ax. 90°, the equivalent sphere is 1 + ½ of 2 = +2.0D. A +2.0D. sph. will put the eye in a condition so that the vertical meridian will be 1.0D. myopic and the horizontal meridian will be 1.0D. hyperopic.

For the most efficient use of the cross cylinder it is best to start the text with the equivalent sphere before the eye.[1]

But how can you find the equivalent sphere?

There are several ways. One effective method is the one suggested by Dr. Williamson-Noble.[2] This is to find the equivalent sphere by a preliminary use of the duochrome test. The spherical lens which equalizes the red and green targets is the equivalent sphere. An understanding of the optics underlying this method will help to visualize more effectively the procedure involved.

Assume a case of astigmatism of 1.0D. with the rule, that is, greater refractive power at or near the vertical meridian. The two focal lines of Sturm's conoid for green light are shown as A and B, Figure 1. They are 1.0D. apart, about one-third mm. This is

the *astigmatic* interval corresponding to 1.0D. of astigmatism.

Between the focal lines is the interfocal circle marked G. The two focal lines for red are C and D, also 1.0D. apart, corresponding to the astigmatic interval of 1.0D. Between C and D is the red interfocal circle marked R.

A and C, as well as G and R, and B and D are separated by 1.0D. (about one-third mm.) corresponding to the *chromatic* interval of 1.0D. For red and blue the chromatic interval usually taken is 1.5D. For red and green it is more nearly 1.0D. and is so generally taken.

If the retina is in the plane B-C, the red and green target will be about equally clear or equally unclear. The green image will be made up of vertical focal lines, while the red image will be made up of horizontal focal lines. This will react somewhat differently on different letters, depending on the importance of their vertical or horizontal strokes. To avoid this, the duochrome test, at least for this purpose, is best made by using the Landolt broken ring, or circular targets.

The yellow for which the eye tends to focus when white light falls on the retina has its focal lines x and y opposite G and R, B and C. Thus the sphere which equalizes the clearness of the red and green target places the eye in meridional balance for the ordinary black and white test chart. The interfocal circle of the yellow is then on the retina, and its focal lines are on opposite sides of the retina.

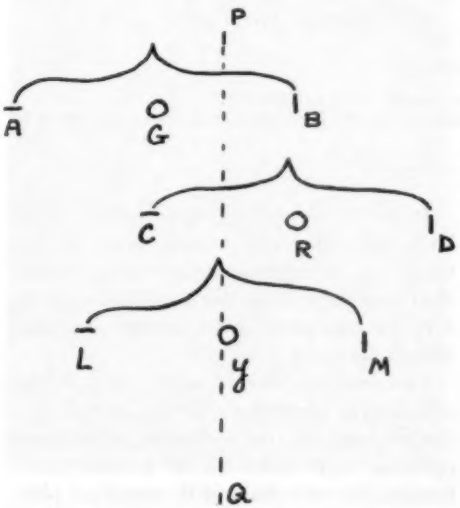

Fig. 2 (Pascal). Astigmatic interval, 2.0D. Chromatic interval, 1.0D.

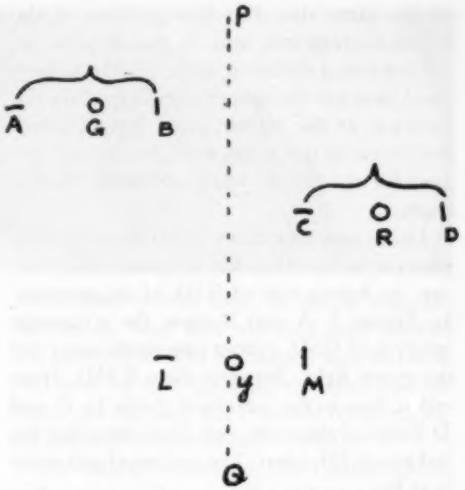

Fig. 3 (Pascal). Astigmatic interval, 0.5D. Chromatic interval, 1.0D.

Let us now assume an astigmatic interval greater than the chromatic interval, say, the astigmatic interval is 2.0D. Then in Figure 2 we have for the green light A and B separated by 2.0D. (about two-thirds mm.). A and C as well as G and R and B and D are separated by 1.0D. corresponding to the chromatic interval of 1.0D. The focal lines and interfocal circle for the yellow are shown at L, y, and M. L is half a diopter from A, y is half a diopter from C, M is half a diopter from R, and so on for other points.

In the duochrome test, the retina lying in the plane half a diopter between G and B, and between C and R, as shown by dotted line P-Q, will have the red and green targets equally clear. The green image will be made up of green vertical ovals, while the red image will be made up of red horizontal ovals

of the same size. But this position of the retina corresponds also to the location of the interfocal diffusion circle for the yellow. Thus here too the sphere which equalizes the clearness of the red and green images places the retina in the position of meridional balance for the regular black and white Snellen chart.

Let us now take a case when the astigmatic interval is less than the chromatic interval, say, we have a case of 0.5D. of astigmatism. In Figure 3, A and B show the astigmatic interval of 0.5D. (about one-sixth mm.) for the green light. Between them 0.25D. from either line is the interfocal circle G. C and D likewise show the two focal lines for the red rays 0.5D. apart. The red interfocal circle is at R.

A and C as well as G and R and B and D are separated by 1.0D. corresponding to the chromatic interval of 1.0D. Half a diopter from A is L, the first focal line for the yellow, in line with B. Half a diopter further is M, the second focal line for yellow, in line with C. When the retina is a quarter diopter from B and from C, as shown at P-Q, the green and red images will be equally clear. The green image will consist of vertical ovals, the red image will consist of horizontal ovals of the same size. In this position again the retina will have on it the interfocal circle of the yellow and the sphere which equalizes the red and green targets on the duochrome test will be the equivalent sphere for the regular chart.

Summary

Cross cylinder tests are most effective when all through the test the eye is maintained in a condition of meridional balance. In this condition the two focal lines of Sturm's conoid are on opposite sides of the retina and at an approximately equal distance.

The sphere which produces meridional balance in a case of astigmatism is called the equivalent sphere. It is here shown that the spherical lens which produces equality of the green and red targets on the duochrome test is the equivalent sphere.

The optics involved are analyzed for three possibilities: (1) When the astigmatic interval is equal to the chromatic interval, (2) when the astigmatic interval is greater than the chromatic interval, and (3) when the astigmatic interval is less than the chromatic interval.

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CARCINOMA OF THE CONJUNCTIVA

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Carcinoma of the conjunctiva has not been described. In other regions of the body, the diagnosis of the carcinoma is being made with increasing frequency. This is a result of the widespread acceptance of exfoliative cytology as a reliable means of diagnosis of carcinoma of the internal organs in its earliest stage.

In the visible and exposed portions of the body, any suspicious lesions should be excised for microscopic examination rather than treating it by methods which do not afford the assurance of a histologic examination of the lesion.

Carcinoma as herein used refers to the microscopic appearance of the surface epithelium whereby the individual cells in the epithelial layer show loss of normal stratification, loss of cellular differentiation, pleomorphism, hyperchromatism—all changes found in malignancy, except invasion.

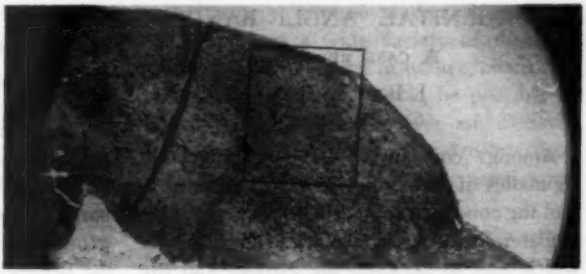

Fig. 1 (Wong). Microscopic picture of the entire lesion. Note the change in the cellular architecture of the central portion of the epithelium as compared to that of the right (fig. 2) (photomicrograph by Dr. F. E. Mohs).

CASE REPORT

A 30-year-old man was first seen on October 13, 1950, with the complaint of redness of the nasal conjunctiva of two weeks' duration.

Examination revealed an elevated, faintly pink pinguecula with a small pigmented spot in its center. This pigmented spot measured a little less than a millimeter in size. It appeared very much like an imbedded foreign body of the conjunctiva but, under the slit-lamp, this was readily ruled out.

The patient was seen on November 3, 1950, three weeks later, at which time the pinguecula was still elevated and showed a pinkish flush. The pigmented spot was now two mm. in size. Surgical excision was advised and the area was removed by an elliptical incision on November 4, 1950.

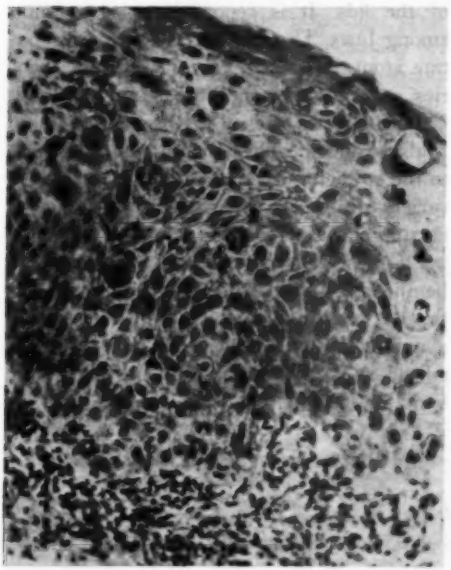

Fig. 2 (Wong). High-power view of blocked-out area in Figure 1. Note the loss of normal stratification and the mitotic figures (photography by Dr. I. L. Tilden).

The biopsy report by Dr. Harold Civin, pathologist at Queen's Hospital is as follows:

Gross. The specimen consists of a soft, fibrous portion of tissue measuring 0.4 by 0.3 by 0.1 cm. There is a flat, black circular area present in its center measuring 0.1 cm.

Microscopic. Histologic examination of a portion of conjunctiva reveals a layer of stratified squamous epithelium, parakeratosis, dyskeratosis, and a considerable amount of variation in nuclear staining properties and size.

Some of the nuclei are large and stained darkly.

Some of the cells show clear spaces adjacent to their nuclei and many nuclei reveal a prominent nucleolus. There are several spaces which show accumulation of acidophilic fluid.

There are numerous blood vessels in close relationship and a marked, chronic inflammatory reaction throughout the tissue, as well as some myxomatous change in the tissue beneath the stratified squamous epithelium.

Diagnosis. Squamous cell carcinoma, Grade I.

When the patient was seen in January, 1952, 15 months later, the area was well healed and no conjunctival abnormalities were noted.

1221 Victoria Street (34).

CONGENITAL "ANGLE BAND"

A CASE REPORT

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Among congenital and developmental anomalies of the conjunctiva, I have failed to find the condition reported in this paper. It is a bilateral subconjunctival fleshy growth over which and in between the growth and the outer angle stretches a white fibrotic band (more marked in the left eye). The growth must partially obstruct the veins as they seem to be congested.

When the patient first came to me, she complained only of headache and eyestrain but, on questioning, she admitted a dragging sensation in her left eye on extreme lateral movement to the right.

CASE REPORT

Miss R. D., a Hindu, aged 17 years, complained of dragging sensation in her left eye on extreme lateral movements. There was no history of trauma or inflammation in the eyes.

Fig. 1 (Bose). Left eye, showing angle band.

Examination. Left eye: A subconjunctival fleshy growth was seen at the outer aspect of left eye. There was a fibrous-looking, white band connecting the outer angle with the growth; two congested veins advanced from the limbus toward the growth (fig. 1).

Right eye: A similar conjunctival growth and congested veins, but no bands, were seen. Both eyes showed the cornea to be normal; anterior chamber, normal; pupil reaction, good; media, clear.

Vision was R.E., 6/6 and with a −0.25D. cyl. ax. 180° = 6/5; L.E., 6/9 and with a −0.25D. cyl. ax. 180° = 6/6.

Fundi oculi were normal.

DISCUSSION

I have had no personal experience with epitarsus, that is, apronlike folds of conjunctiva attached to the inner tarsal surface of the lids. It is reported to be common among Jews. Though epitarsus is quite separate from the present case, its etiologic theories may fit this condition herein reported.

The theories are (1) Presence of these folds is due to adhesion of amniotic bands to the epithelial covering of the globe, (2) a variety of plica semilunaris; (3) due to intrauterine conjunctivitis.

Of these the first theory seems to me to be the most probable, and fits in with the present condition. The condition seems to be a new entity and perhaps has not been reported so far. The name "congenital angle band" is suggested.

223 Bowbazar Street (12).

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ACUTE GLAUCOMA IN PATIENT WITH PEPTIC ULCER*

Case report on use of a new antispasmodic, Bentyl hydrochloride®

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Fredericksburg, Texas
and
G. D. Guilbert† M.D.
Kerrville, Texas

The danger of precipitating glaucoma with orally administered drugs with atropinelike action has been emphasized by Ullman and Mossman[1] who found an incidence of 0.75 percent exacerbation of glaucoma in patients administered this type drug. The use of the atropine-type drugs (both the natural alkaloids and synthetic substitutes of a similar nature) as antispasmodics for gastro-intestinal disturbances of functional and pathologic origin necessitates caution and extreme care in patients with a history or symptoms suggestive of glaucoma or preglaucoma.[2]

The action of these drugs is on the autonomic effector cells and the intraocular pressure may be elevated by the mechanism of this action. The need of a drug for patients with chronic glaucoma or preglaucoma symptoms who have or develop concomitant gastro-intestinal disturbance of a serious nature is apparent.

A potent, selective antispasmodic, Bentyl® hydrochloride, was described by Brown, Thompson, Klahm, and Werner.[3] In animals it has about one-eighth the milligram potency of atropine when assayed against gastro-intestinal motility, whereas it is necessary to administer about 500 times the atropine dose systemically to dilate the pupil.

Preliminary clinical studies in patients suffering from a wide variety of diseases, largely gastro-intestinal in nature, confirmed the hypothesis that it would be possible to administer an effective dose of Bentyl® which would restore normal gastro-intestinal motility without producing the usual atropinelike side effects.[3-9]

In a comparison of Bentyl® with other natural and synthetic alkaloids, there were 163 patients who received the drug parenterally, even in dosage as high as 100 mg. (five times the usual oral dose) without producing mydriasis.[10] Accordingly, the drug was used in the following case in which antispasmodic therapy was indicated as part of the peptic ulcer management of a patient, who had also developed acute glaucoma.

Case history

B. B., a 73-year-old white man, was admitted to Kerrville Veterans Hospital, in 1945, with a diagnosis of pulmonary tuberculosis, emphysema, and arteriosclerosis. Under treatment the pulmonary tuberculosis became quiescent. The patient developed a peptic ulcer in 1946. The peptic ulcer became inactive under treatment with antacids, diet, and tincture of belladonna as an antispasmodic.

On July 6, 1951, the patient developed symptoms of glaucoma in the left eye and an Eye, Ear, Nose, and Throat consultation was requested.

Examination of the eyes revealed in increased intraocular pressure in the left eye of 66 mm. Hg (Schiøtz).

Initial treatment consisted of introducing Mecholyl® crystals into the conjunctiva of the left eye which reduced the intraocular pressure to 58 mm. Hg (Schiøtz) in 15 minutes. Following the use of Mecholyl® crystals, two-percent pilocarpine combined with 0.5-percent eserine drops was used every 15 minutes for four hours and then hourly for 24 hours.

Two hours and 15 minutes after treatment of the glaucoma was started, the patient de-

* From the Eye, Ear, Nose, and Throat Service, Veterans Administration Hospital, Kerrville, Texas. Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

† Now stationed at the Veterans Administration Hospital, Waukesha, Wisconsin.

veloped a gastric crisis with vomiting of large amounts of clotted blood. This complication was controlled with intravenous 0.5-percent procaine.

On July 7, 1951, the intraocular pressure was reduced to 37 mm. Hg (McLean) and the patient had no discomfort in the left eye, but continued to complain of gastro-intestinal discomfort.

The eye drops were changed to Carcholin® (1.5 percent) combined with pilocarpine (two percent) and Zephiran® (1:5,000). On August 30, 1951, it was decided to try the patient on Kolantyl® tablets (Kolantyl® contains 5.0 mg. Bentyl hydrochloride per tablet). He received one tablet per day as a substitute for the tincture of belladonna he had received for the peptic ulcer. The intraocular pressure remained normal throughout the period of treatment with Kolantyl® which was discontinued on September 25, 1951.

On January 4, 1952, the patient had an exacerbation of acute glaucoma in the right eye. Tension was: R.E., 70 mm. Hg; L.E., 50 mm. Hg (McLean). This was controlled by miotics which were instilled three times daily. The patient's intraocular pressure has remained normal since that time.

On April 22, 1952, Bentyl® capsules (10 mg. each) were given three times daily to check its effect on the intraocular pressure. Before treatment the tension was: R.E., 25 mm. Hg; L.E., 40 mm. Hg (McLean). At the end of one week's treatment, April 29, 1952, the tension was: R.E., 27 mm. Hg; L.E., 36 mm. Hg (McLean). The drug was then discontinued.

Conclusion

1. The safe oral use of Bentyl hydrochloride® in a patient suffering concurrently from peptic ulcer and acute glaucoma is described. The drug did not appear to influence the intraocular pressure, or the course of acute glaucoma, when given to this patient in the dosage commonly employed in the treatment of gastro-intestinal disorders.

2. The patient's intraocular pressure remained normal while given a trial of Bentyl® in full therapeutic dosage.

3. This case is reported in the hope that further investigation will confirm, or disprove, these findings.

4. These findings do not cover an extended period of time and further investigation will reveal whether the Bentyl® will have an accumulative effect causing a rise in intraocular pressure after prolonged use. The findings from the results reported in this paper indicate an actual lowering of tension which is assumed to be due to the improvement in the patient's general condition.

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VISION IN INDUSTRY

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Medicine found its way into industry with the advent of the compensation laws in the various states, beginning about 1910.[1] Regulations for head and eye protection for the State of Pennsylvania were made by the Industrial Board of the State and were made effective July 12, 1921.

The Pennsylvania Workman's Compensation Law provides that compensation shall be payable at the rate of 66⅔ percent of the weekly wage, not to exceed 30 dollars per week, during a period of 125 weeks, for the loss of or industrial loss of use of an eye due to an accident while in the course of employment.[2]

Kuhn's[3] book states that the appraisal of loss of visual efficiency was decided by a Committee of Ophthalmologists of the American Medical Association in New York, June 10 to 14, 1940. This committee decided that no change need be made from the report of a committee of 1925 except for one revision:

In the report of May, 1926, a recommendation was made "that compensation status be so changed that awards for ocular disability shall be based on the percentage of permanent disability only," that is, on the percentage loss of both eyes.

This recommendation is now omitted and it is suggested in this revision that the present common statutory method be followed—that is, that the method for determining the percentage loss of vision be based on the loss of efficiency of one eye, and that disabilities involving both eyes be based on a weighted method of compensation.

As to the regulation for computing compensation the following in part was also taken from Kuhn's[3] detailed analysis of the Ophthalmology Committee Report of the American Medical Association, 1940:

Final examination, upon which compensation is to be based, shall not be made until at least three months shall have elapsed after all visible evidence of inflammation have disappeared, except in cases of disturbance of extrinsic ocular muscles, optic-nerve atrophy, sympathetic ophthalmia, and traumatic cataract; in such cases, 12 months and preferably not more than 16 months shall intervene before examination shall be made on which final compensation is to be computed. In cases of additional loss in visual efficiency when it is known that there was a pre-existing subnormal vision, compensation shall be based on the loss incurred as result of eye injury or occupational condition specifically responsible for additional loss. If no existing records are present, it shall be assumed visual efficiency prior to injury was 100 percent.

According to the information supplied by the Secretary of Labor of Pennsylvania, there was found to be no definite law pertaining to the visual requirements for workers in the industries of the State of Pennsylvania.

The reason for this is self-explanatory when one thinks of the many variances of vision required to do different jobs. For example, a machinist grinding fine tools requires good close vision and binocular vision, while a janitor or delivery boy may do his job with less qualification. Thus, a man may have subnormal vision and still be excellent in the type of work he is doing.

The arbitrary figure, which most oculists accept as industrial vision, is vision which is better than 20/70. Berens[4] defines "economic blindness" as meaning a central visual acuity of 20/200 or less in the better eye with correction; or central visual acuity of more than 20/200, if there is a field defect in which the peripheral field is so contracted that the widest diameter of the visual field subtends an angular disturbance no greater than 20 degrees.

METHODS OF TESTING VISION IN INDUSTRY

Oculists feel the simplest way to take vision is by the Snellen wall chart. The lighting on the chart should be 10 to 20 foot-candles and evenly distributed over the chart; the room lighting should be one fifth as bright as the chart light. One must be sure

a complete cover is used alternately before each eye. Different letters should be used for each eye. A reading card should be used each time for noting the amount of accommodation present, in other words to note if bifocals or reading glasses are needed for close work. Both pre-employment and periodic visual checking should be done. A complete examination of vision should include vision check for distance and near, steropsis, and muscle balance.

Visual efficiency must be included in visual requirements and Vail[5] clearly summarizes the factors affecting visual efficiency into the following groups: (1) Visibility; (2) the general health of the worker; (3) distance and near visual acuity; (4) visual field; (5) ocular motility; (6) color vision or sense; and (7) injury or disease of the cornea.

When one talks about visual efficiency, two very important factors which most of us neglect to think of in detail are illumination and color. So often many of us will check the vision of an individual without realizing where the person uses his eyes.

Birrin[6] reminds us that the lighting engineer and physicist have adopted the same views and methods on illumination. For example, the lighting engineers agree universally that high levels of illumination and light colors are desirable for efficient seeing. The gestalt psychologist in simple terms does not agree with this; the explanation in gestalt psychology is that where there is a strong light or high brightness, the human organisms will seek equilibrium and balance. High brightness in the field of view, if isolated from the task, may cause disruption.[6]

One must not forget that sounds have variance of effect on the perception of color and on the vision itself. Loud sounds decrease the rod sensitivity of the eye, while increasing the sensitivity of the cones.[7] This same phenomenon is proven more by the fact that sensitivity to red colors is lowered by influence of sound, while sensitivity to green colors is increased.[8] As to working conditions in relationship to color around the worker, one must remember that time is overestimated under red light and underestimated under green light.[9]

The salient facts to be kept in mind as far as vision in industry is concerned are:

1. Compensation results should not be decided too hastily.

2. An employee may have subnormal vision and still be excellent in the type of work he is doing.

3. Although there is no definite law, better than 20/70 vision is considered industrial vision by most oculists.

4. The simplest method for taking vision is by the Snellen wall chart.

5. Visual efficiency must be included as a part of visual requirements.

6. The place where the employee works must not be forgotten when visual efficiency is considered.

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SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

December 1, 1952

DR. ABRAHAM L. KORNZWEIG, *President*

MARK J. SCHOENBERG MEMORIAL LECTURE

PROGNOSIS AND TREATMENT OF RETINAL DETACHMENT

DR. C. L. SCHEPENS said that the most important elements of prognosis depend on certain characteristics of the retinal breaks and on the location and extent of fixed retinal folds. There are five different types of the latter: circular folds, star-shaped folds, meridional folds, massive vitreous retraction, and folds associated with visible retinitis proliferans. Prognosis in total retinal detachments and in long-standing detachments was discussed. The chance of good functional recovery is dependent upon factors such as the existence of macular cyst, macular break, and postoperative macular degeneration of which there are three morphologic types.

Surgical treatment of retinal detachment cannot be discussed in detail. Some of the points stressed in this paper are: anesthesis, keeping the cornea clear and the pupil dilated and dissecting the sclera carefully, localization of the breaks, and application of surface diathermy. The dangers of using many perforations was stressed. An attempt was made to evaluate the postoperative fundus picture on the operating table.

In fairly favorable cases, the postoperative course may either be one of steady improvement or residual subretinal fluid may be present; treatment for the latter case was discussed. Cases in which untreated breaks are discovered postoperatively and cases showing fixed folds preoperatively create special problems. Postoperative choroidal detachment is a frequent cause of error in evaluating a postoperative situation.

Scleral buckling is particularly indicated in cases in which the ordinary type of retinopexy has little chance of being successful, such as in cases with massive vitreous retraction, large fixed folds, visible strands pulling on the retina, breaks showing rolled-up edges, giant breaks, organized blood in the vitreous, puckering of the ora serrata, and detachments occurring after extraction of an intraocular foreign body. This procedure is also indicated in complicated cases showing one of the following features: total detachment, aphakia, reoperation, long-standing detachment.

The procedure of scleral buckling resembles that of lamellar resection, described by Paufique, with the following important differences: careful preoperative examination with a stereoscopic indirect ophthalmoscope, placing the buckle on the disc side of the retinal breaks in spite of all technical difficulties, and adding no diathermy application of any kind on the disc side of the buckle.

One-hundred scleral-buckling operations were performed on 85 eyes between January and October, 1952; 74 cases were followed for at least two months after operation; of these, 65 percent showed reattachment of the retina. This compares favorably with similar statistics already published, but it is too soon to pass final judgment on the value of the procedure.

BERNARD KRONENBERG,
Recording Secretary.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

SECTION ON OPHTHALMOLOGY

December 18, 1952

DR. GEORGE F. J. KELLY, *chairman*

BULLOUS KERATITIS AND EPITHELIAL CORNEAL DYSTROPHY

DR. HAROLD D. BARNSHAW presented a man, a clerk aged 45 years, who was first seen in March, 1949. He complained of feeling a bubble on the left eyelid, accompanied by slight pain. These symptoms had been present for approximately three weeks. During this period he had been refracted and new glasses ordered. His ocular and general medical histories were negative.

Examination of the right eye revealed a small scar of the cornea at the 6-o'clock position close to the limbus. This was superficial and did not penetrate deep into the cornea. There were a few fine pigment deposits on the posterior portion of the cornea. Tension was normal.

The left cornea revealed an edema in the inferior temporal quadrant with lowered sensitivity. The slitlamp showed this to be in the epithelial layer. There were a few fine pigment deposits on the posterior portion of the cornea. Tension was soft. The pupil was dilated with homatropine. At this time, vitamin A, two-percent Dionin, and hot compresses were ordered.

Two days later, there was a large single bulla in the inferior temporal portion of the left cornea, measuring 1.5 mm. in diameter. The epithelial portion of the bulla was clear, as was the base. The bulla was removed with a cotton applicator, and the base touched with tincture of iodine.

Since then, the corneal changes have advanced in spite of all treatment. He has been subjected to tincture of iodine and trichloracetic acid locally. Atropine, Dionin, zinc, holocaine, sulfacetimide, and cortisone drops, and cod-liver ointment have been used. Hot compresses and pressure bandages were also used. In addition, superficial keratectomy was tried.

In April, 1949, the patient was seen in consultation by Dr. Alfred Cowan. Dr. Cowan's diagnosis was idiopathic bullous keratitis. His consultation reports are given here in full:

"On April 23, 1949, the right eye was negative for the purpose of this report.

"In the left eye, grossly, the conjunctiva of the lids and of the globe was intensely injected. There was considerable ciliary injection. The lower half of the cornea was irregularly infiltrated, the surface unevenly elevated, and with a large white bleb below.

"With the biomicroscope, the epithelium was edematous in varying degrees over the entire cornea. The epithelium over almost the entire lower quadrant was thickened, considerably relucent and elevated, forming a vesicle containing clear fluid. The base of the vesicle, the anterior surface of Bowman's membrane and the zone immediately behind were intermittently increased in relucency and irregularly refractile.

"In the upper portions of the cornea, there were a number of small vescicles, some with thickened epithelial surfaces. The epithelium was eroded in several places. But mostly the edema of the epithelium over the upper part of the cornea produced only a bedewing. The stroma was irregularly relucent. There were no blood vessels in the cornea proper.

"Well-advanced endothelial dystrophy and numerous folds and ruptures in Descemet's membrane were present, but no other evidences of uveal involvement were seen.

"On June 29, 1949, the condition was essentially the same, but with a large area of denudation of epithelium in the center of the cornea.

"On October 4, 1952, there was only slight ciliary and conjunctival congestion. The right eye was still practically negative. In the left eye, the epithelium was edematous over its entire surface, varying in degree from a moderate bedewing to dense bullas. The

cornea was irregularly infiltrated throughout its entire thickness with scattered, comparatively lucid areas. The portions of the posterior surface of the cornea which could be made out were characteristic of guttate cornea, indicating entire disintegration of the endothelium.

"The anterior chamber was empty; the iris, as well as could be seen, was intact and active, and the lens was about normal."

After Dr. Cowan's examination in October, he felt that the patient had an epithelial and endothelial dystrophy of Fuchs.

At the present time, the patient's vision is 6/9 in the right eye and 6/30 in the left eye. The right eye shows little except some changes in the endothelium. The left eye shows the marked epithelial changes with superficial scarring, and many small bullas of the lower two thirds of the cornea. The endothelial changes are marked.

This case is more representative of epithelial dystrophy of Fuchs than of any other form. Fuchs first described this condition as an entity in 1910. He characterized it as: (1) The disease attacks elderly persons and is slowly progressive; (2) no inflammatory symptoms accompany the progress of the lesions; (3) there is a diffuse opacity of the cornea most intense in the central region shading off to a clear zone in the margin; (4) the corneal epithelium is roughened, deficient in luster, and contains numerous vesiclelike elevations.

Fuchs reported 13 cases, four in men and nine in women. He noted that it was frequently associated with glaucoma but did not follow glaucoma. Dr. William Zentmayer, in 1918, reported a case of epithelial dystrophy of the cornea, bilateral, in a woman, aged 65 years. In 1926, H. and J. S. Friedenwald suggested that the endothelial changes were probably the primary phenomena in typical cases of epithelial dystrophy.

This patient is young for this disease, most persons having it being well over 50 years of age. The edema, which usually starts in the central area, began in the periphery in this case. However, definite epithelial degenerative changes are shown, as marked by edema, bulla formation and stria in the parenchyma. The endothelial changes have not played a prominent role in this patient.

Discussion. Dr. Alfred Cowan:

Bullous keratitis is not a disease entity. It represents an advanced stage of corneal edema. It occurs in many ocular affections, inflammatory, degenerative, or dystrophic. In my opinion this is undoubtedly a case of Fuchs' epithelial-endothelial dystrophy.

The condition which complies with what Fuchs chose to call epithelial dystrophy is not limited to the epithelium. Eventually it involves all the layers of the cornea—the epithelium, the entire stroma, Descemet's membrane, and the endothelium. It is even claimed by many that it does not start in the epithelium but that the disease first attacks the endothelium. You might even infer from much of the literature on the subject that every case of endothelial dystrophy is actually the first stage of the involvement of the rest of the cornea.

I do not agree with those who maintain that endothelial dystrophy, even the most advanced case, is necessarily the early manifestation of eventual dystrophy of the entire cornea.

Fuchs' epithelial dystrophy is rare; endothelial dystrophy is common. I have had under observation for years many cases of endothelial dystrophy that have progressed to disintegration of the entire endothelium—guttate cornea—without the slightest indication of involvement of the anterior layers and with comparatively little loss of usefulness of the eyes.

Neither, in my experience, does the endothelium act as a barrier to prevent fluid from the anterior chamber from entering the cornea, and subsequent edema. The theory that the sick endothelium allows fluid to pass through it does not seem to hold. For years I have seen eyes with total absence of endothelium with never the slightest evidence of edema. Other factors must be

present. Cogan's theory, that the edema and the epithelial changes are due to an osmotic imbibition of fluid from the tears, is much more tenable.

Treatment of opacities by desiccation

Dr. William M. Hart and Dr. Louis F. Hinman presented a paper on "The treatment of opacities of the cornea by desiccation."

In 1946, preliminary observations were presented before this group on the physical and chemical factors affecting the transmission of light by the cornea and sclera.* The observation was made at that time that corneal opacities could be made to disappear in vitro in one of two ways: (1) By simply drying the cornea in air, and (2) by hydration in suitable solutions. The physical reasons for this were given.

At the present time, data are presented on the treatment of interstitial opacities in dogs due to distemper. A thorough desiccation of the cornea with dry air is followed by disappearance of the opacity over a period of three to six weeks. Only a fine interstitial haze remains as a permanent result.

The results of three human cases are also offered at this time—one case of interstitial luetic opacity and two cases of opacity following unsuccessful keratoplasty. These eyes were very debilitated before treatment began so that the objective benefits obtained were of little practical importance in benefiting vision. The desiccation technique, to be described elsewhere, is not offered as a method of therapy at this time but serves only to demonstrate the physiologic potentialities of the opaque cornea.

Discussion. Dr. Glen G. Gibson:

This interesting investigation by Dr. Hart and Dr. Hinman on the effects of partial desiccation of the normal and diseased cornea brings up many points for discussion, I shall mention only a few.

I feel that Dr. Hinman would like to have it pointed out that his role was that of clinical consultant, and the credit for this concept rightly belongs with Dr. Hart.

It has been my privilege from time to time to observe both the patients and the dogs upon whom this procedure was performed, and I should like to congratulate the authors not only for their effort but also for the conservative and accurate nature of their work and their presentation.

The material presented by Dr. Hart and Dr. Hinman was selected with two dominant considerations in mind:

First of all, in an effort to avoid the error of attributing improvement to their work, which was due to the natural improvement of opacities in the cornea, they selected patients whose disease was long standing, well past the possible edematous phase of lesions, in which the permanent nature of the corneal opacities could not be doubted.

The second important consideration was that only patients who had maximum extent and maximum depth of the corneal opacities were selected so as to represent those patients who had, so to speak, nothing to lose by this procedure. They accepted these obvious disadvantages because of the possible potential dangers which might be inherent in a procedure involving such a profound desiccation.

Dr. Hart failed to point out in his description the intense nature of this drying. The cornea was really desiccated in a very thorough manner. The authors were concerned about such dangers as, for example, decreasing corneal immunity with resulting infection, and interfering with the functional integrity of the corneal endothelium. Either incident might add to, rather than subtract from, the amount of corneal opacity. Furthermore, the material which they selected did not represent any one specific clinical corneal entity, but rather constituted a heterogenous group of conditions. Therefore, the

* Hart, W. M., and Chandler, B. F.: The cornea. I. Swelling properties of the fibrous tunic of the eye. Arch. Ophth., **40**:601-612 (Dec.) 1948; II. Factors affecting the transmission of visible light by the fibrous tunic of the eye. Arch. Ophth., **40**:612-623 (Dec.) 1948.

term corneal opacity is used in the widest sense.

We were formerly content to regard corneal opacities merely from the microscopic point of view. We considered that the loss of transparency was adequately explained by cellular and fibroblastic proliferation; the distorted arrangement of the corneal lamellae was secondary to the pathologic process.

It is becoming increasingly evident that it is necessary to develop a more intimate concept of the chemical composition of opacities. The remarkable clearing that occurred in these corneas suggests it may be necessary to modify our former concept. The work here presented suggests that, even in nonedematous corneal opacities, water contributes in a large measure both mechanically and chemically to the derangement of the optical properties of the cornea. Microscopic observations have never led one to suspect these facts.

An honest and accurate evaluation of this work is not only difficult, it is, perhaps, premature. What has been accomplished is this: it has been adequately demonstrated that corneal opacities can temporarily be cleared with surprising ease and speed. This can be done and can be repeated without apparent damaging effect to the eye.

What remains to be done, beyond this first stage of the work, is to demonstrate that the clearing so attained can be maintained in sufficient amount and in a sufficiently lasting form to be of real clinical value. It is to be hoped that, in the future, this method, or some modification of this technique, perhaps to include drying of the posterior surface as well as the anterior, or maybe another method, may be used to control the pathologic processes causing opacities.

It must be clearly understood, however, that this work has not progressed beyond the experimental stage. It is by no means sufficiently effective or advanced to be recommended as an acceptable method of treatment of corneal opacities.

Dr. Francis Heed Adler:

I would like to emphasize, as Dr. Gibson has, that experiments of this sort are brave attempts to explore a field that has been particularly barren, so far, on both the side of clinical medicine and the side of research. Fortunately, we are now in a position where we feel rather well grounded in the belief that Cogan and Kinsey's theories of corneal turgescence and transparency are correct. However, these theories have not been entirely substantiated, and I am sure Cogan and Kinsey themselves would be the first to admit this. So far they are the best that have been presented. We must not stop with these but explore further possibilities.

These experiments show no greater clearing of opacities of the cornea than such a well-recognized agent as glycerine which temporarily clears turgescent corneas. They make us no wiser as to what fundamentally causes the opacification in these cases, but they are an earnest and honest attempt to find the truth; therefore, the authors should be congratulated.

Dr. Louis F. Hinman:

Dr. Gibson and Dr. Hart covered these things very adequately. There are two things that are outstanding to me. I should like to call to the attention of ophthalmologists how fortunate we are that the Lord, and tissue tolerance, keeps us out of a lot of difficulties. These corneas were dehydrated almost to an amorphous mass, and to see them again returned to the semblance of normal was quite a revelation. Secondly, it suggests that perhaps there is some place for stored corneas, rather than immediate transplantation of corneas, for corneal opacities.

At the risk of being a bit of a heretic, I should like to mention my observation concerning vascularization. In several of these cases, prior to improvement in the appearance of opacities, neovascularization was observed. It may very well be that vascularization is not quite the ominous process we have considered it. It may in some way contribute to clearing of the cornea following injury.

Dr. William Hart (closing):

I wish to thank Dr. Gibson and Dr. Adler for their comments. One thing we did not emphasize is the fact that the beneficial effect of this treatment on corneal opacity is a biphasic thing. Acutely, the opacity disappears with desiccation which, superficially, makes it appear that simple withdrawal of water is all that is concerned. The opacity does reappear on rehydration. However, there follows a period during which the clearing effects reappear and become permanent.

This result was a surprise to me. We are all fond of our pet theories. I was so intrigued with the mechanical concept that I had visions of the cornea clearing immediately and staying that way. It only serves to emphasize that the physical-chemical factors have been no more than enumerated. We do not know their quantitative relationships to each other. However, regardless of mechanisms, the encouraging thing from the practical standpoint is that we do not need to regard fibroblastic invasion and scarring as a hopeless situation.

Local anesthetic agents

Dr. Harold G. Scheie and Dr. Richard Ellis presented observations on the experimental and clinical use of Efocaine and a substance designated U-0045, two local anesthetic agents having a considerably longer action than procaine. The advantages which would result from local anesthesia of sufficient duration to avoid postoperative pain in intraocular surgery seem obvious.

Efocaine, a local anesthetic which embodies a new approach to prolonged anesthesia, has recently been introduced for clinical use. It is made by dissolving water-insoluble procaine base in polyethylene and propylene glycol. When this solution is injected into the body, upon contact with tissue fluids, the procaine crystallizes out of solution to form an anesthetic depot which requires a week or more to absorb. The anesthetic remains effective during this time.

Still more recently, a local anesthetic agent designated U-0045 (Upjohn) was made available to us by our Department of Anesthesiology. U-0045, Bis-[B-(o-methoxyphenyl)-isopropyl]-amine, differs chemically from the procaine group of anesthetics in that it is a secondary amine and contains no ester linkage as does the procaine group.

Because of this structure, it is less susceptible to hydrolysis by tissues and therefore has a prolonged anesthetic action as compared to procaine. Like procaine base, it is insoluble in water, but the salt, U-0045 lactate, which was used in our work is soluble up to five percent.

Bass and Schroeder found it to be four times as toxic as procaine, but it is 40 times as effective an anesthetic agent. This ratio gives it a therapeutic advantage of nearly 12 times so that it can be used with safety.

We compared the effect of retrobulbar injections of procaine, U-0045, and Efocaine in rabbits.

Procaine was found to be effective for an average of 60 minutes but, when combined with hyaluronidase, its effect lasted only 33 minutes. The shortened duration of action when combined with hyaluronidase was probably due to the more rapid diffusion through the tissues with more prompt elimination of the local anesthetic.

U-0045 lactate maintained an effective block of the ciliary nerves for an average of 312 minutes but, when combined with procaine, this period of effectiveness was reduced to 258 minutes. The anesthetic effect of Efocaine was still more persistent, lasting for 11 days or longer in nearly all instances.

In rabbits, U-0045 caused very little local reaction compared to Efocaine which was extremely irritating. Retrobulbar injections of Efocaine caused rather marked proptosis and conjunctival edema. Anterior-chamber injection caused permanent corneal opacity with corneal vascularization. Intracorneal and subconjunctival injections were likewise extremely irritating as compared to U-0045.

Clinically, Efocaine produced effective retrobulbar anesthesia varying from one week to two months. The drug caused so

much reaction in the six patients in whom it was employed, however, that we discourage its clinical use. Exophthalmos and edema of the lids always occurred. One patient developed weakness of the superior rectus muscle with ptosis which lasted for a period of two months.

Our clinical experience with U-0045 was more encouraging. When instilled into the conjunctival cul-de-sac, a 0.2-percent solution produced corneal anesthesia for a longer time than did 1.0-percent pontocaine. It caused more local irritation in patients but produced no damage to the corneal epithelium.

It was used for retrobulbar injection in over 100 patients for various types of intraocular surgery. U-0045 lactate alone produced excellent anesthesia lasting from seven to 12 hours.

Akinesia of the extraocular muscle was not as satisfactory as the akinesia that we had been obtaining with procaine. We, therefore, combined U-0045 lactate with procaine in the same solution and used this combination in 95 patients. The anesthesia was universally good and the akinesia of extraocular muscles excellent. The anesthesia, as a rule, lasted for eight hours and consequently postoperative pain was greatly diminished.

Only an occasional patient complained severely enough to need an analgesic and postoperative vomiting was almost elmiminated. We feel that this anesthetic agent represents a real advancement in the postoperative management of patients who have had ocular surgery.

Discussion. Dr. James Eckenhoff:

For years there has been an attempt to increase the comfort and safety of the patient during operation and in the postoperative period by the employment of drugs preoperatively. You are all familiar with the once-popular use of basal narcotics to increase the patient's comfort before, during, and following operation, and to decrease the incidence of complications. As you know this method decreased rather than increased the safety of the patient, so it has generally been abandoned.

Along the same lines, local anesthetics have been injected during or immediately following ophthalmic surgery for the purpose of relieving pain. Here again, they have not been uniformly successful and, for the most part, have been abandoned.

Two years ago, we were approached by a detail man trying to sell us Efocaine on the basis that it was a drug that could be used for nerve blocks the night before operation, the next morning the patient would have good anesthesia for his operation, and little pain for eight to 10 days postoperatively.

It sounded attractive but somehow impractical. However, we did try the drug for a large number of nerve blocks, and have obtained a considerable experience with it.

Blocks have been satisfactory in about 75 percent of the patients, but complaints in the remainder of the cases have made us abandon the drug. Complaints have been of pain upon injection; pain in areas adjacent to that blocked, for example, pleuritic pain following stellate ganglion block; and pain in blocked areas occurring on disappearance of the anesthesia.

About eight months ago, Dr. Scheie asked our opinion of the use of Efocaine for ophthalmic surgery. With our experience, we were not particularly enthusiastic, and said that, although some approved of the drug, we advised caution. The results Dr. Scheie has shown tonight verify our opinion.

About this same time we were working with Orthoxin, and suggested that Dr. Scheie might like to employ this drug. We had performed about 100 regional blocks with it, and found it to be an effective anesthetic. Onset of the block seemed to be slower than with procaine, but the duration of anesthesia was considerably longer, as long as four or five hours. Interestingly enough, in view of the difference presented by Dr. Scheie—objective signs of block for 47 minutes and subjective signs for 77 min-

utes—many of our patients had subjective relief of pain for hours after the block had apparently worn off.

We have found one toxic effect of U-0045 and that has been hypotension. We employ, however, considerably larger quantities of the drug (25 to 40 cc.) than are used in ophthalmic surgery. I would like to ask Dr. Scheie if he noticed any toxic symptoms with this drug?

One thing that worries me is that we have employed this drug intrathecally in 85 patients. The anesthesia has been uniformly satisfactory, and we have seen no postspinal neurologic sequelae. I believe, though, in view of the tissue reaction that Dr. Scheie observed following instillation of the drug into the conjunctiva that we probably should abandon the use of the drug for spinal anesthesia pending further study. We went ahead with the intrathecal injections following satisfactory results in a large series of animals.

Finally, I would like to ask Dr. Scheie if he has attempted to explain why Efocaine is a tissue irritant? After all, it is procaine yet it does irritate tissue.

Dr. Francis Heed Adler:

When Dr. Scheie first talked to me about U-0045 and its possibilties, I said, "Well my patients don't have any pain after procaine injection," and then he asked me, "Well, do you come around and see them three or four hours after surgery?" I said, "No."

The way to evaluate a patient's postoperative pain is to ask your resident about it. They are much more comfortable, according to our residents, when we use U-0045 than when we use procaine. I feel certain that the anesthesia lasts longer, and that they are more comfortable. When I come around, and ask them whether they have any pain, they all say "No," but then that is next day.

I do think it takes somewhat longer for this anesthetic to act, and I would like to ask if this is not true, when you inject U-0045, do you not have to wait longer than when you inject procaine for the anesthetic to take full effect?

Dr. Solomon S. Brav:

Has Dr. Scheie employed this drug for akinesia—as an O'Brien block—or just as a retrobulbar block?

Dr. George F. J. Kelly:

Have either one of these drugs been used in patients who already have pain? Most of the cases that you have talked about are those in which the pain is postoperative in type. Do you know whether they have been used by the neurosurgeons for relief of pain such as occurs in tic douloureux?

Dr. James Eckenhoff:

There has been a good deal of work with the drug for relieving pain. Fifty to 60 percent of our own experience has been in the treatment of chronic pain of varying causes. It has been employed for relief of intercostal neuralgias, for sympathetic blocks to relieve autonomic dystrophies, and I believe it has been tried for tic. It has been employed by others to relieve pruritus ani, with the injection of occasional enormous doses. One author reported the use of 15 cc., and then was surprised when the patient developed a rather wide slough. The results have been fairly much as already indicated.

Dr. Harold G. Scheie (closing):

Dr. Ellis and I wish to thank Dr. Eckenhoff and the other discussors for their interesting comments and questions.

I would like to point out the significantly longer duration of anesthesia in patients than in our animals. This probably resulted from the use of one cc. of 0.5-percent solution in patients as compared to 0.5 cc. of 0.2-percent solution in animals. We occasionally found that anesthesia lasted for over 12 hours in patients, a sufficiently long period to avoid nearly all postoperative pain.

In answer to Dr. Eckenhoff's question, we have observed no toxic symptoms from the amount of U-0045 which we have used. I should think this substance would be used intrathecally for spinal anesthesia only with

hesitation because of irritation, as shown by conjunctival instillation, and, probably more important, because anterior chamber injection caused corneal haze in five of 11 eyes.

Dr. Adler has mentioned the incidence of postoperative pain following intraocular surgery. We find it occurs for four to six hours in most instances. Our nurses are very grateful for U-0045, because, by avoiding this period of pain, their job is made much easier and their problem of postoperative care is much more simple.

Dr. Brav asked if we used it for facial akinesia. We have not because the duration of akinesia from procaine is always sufficiently long.

M. Luther Kauffman,
Clerk.

CHICAGO OPHTHALMOLOGICAL SOCIETY

October 6, 1952

Dr. William F. Hughes, Jr., *president*

Clinical meeting

Presented by the Department of of Ophthalmology, Northwestern University Medical School

Macular degeneration

Dr. Walter Hagens presented a white man, aged 63 years, who had worked as a painter. Vision was normal until April, 1941, when it was suddenly lost in both eyes. Sight returned slowly after about 20 minutes but never to normal. There was no vertigo nor loss of consciousness. Since that time, there have been numerous similar episodes, lasting only several seconds or minutes.

There are no associated cerebral signs but it is believed that the attacks may represent a type of petit mal and may be only indirectly related to the fundus changes. Prior to onset of the ocular symptoms he was a heavy cigarette smoker.

For many years he has had intermittent claudication; X-ray studies of the legs and feet reveal extensive calcification of the vessels. The medical diagnosis is arteriosclerosis obliterans.

Serology and chest films were negative. Physical examination revealed moderate hypertension, hepatomegaly, and lack of arterial pulsations in the extremities.

Vision was limited to counting fingers at two feet; a dense central scotoma extending out 10 to 15 degrees from fixation was present in each eye. External examination was negative. Tension was normal. The ocular medias were clear, the discs showed slight peripapillary choroidal atrophy. The arterioles were moderately attenuated and there were no hemorrhages or exudates.

In each macula, there was a slightly elevated gray-white area of scar tissue over which the retinal vessels coursed. Surrounding this area, the retinal pigment was thin and the choroidal vessels were easily seen. A few clumps of pigment were scattered over the lesions and also in the periphery of the fundus.

Although the fundus at this time does not resemble that described by Gifford and Marquardt and the patient is older than the group described by them, it was stressed that this case may well represent the terminal picture of vasospasm involving the perifoveal retinal vessels, especially when the evidence for peripheral vascular spasm is considered.

The absence of marked pigment proliferation and vitreous opacities would speak against an inflammatory process, and the early age of onset and lack of significant elevation or hemorrhagic extravasation at the periphery makes Kuhnt-Junius disease unlikely. The sudden onset and glial tissue proliferation are not characteristic of the primary change in senile macular degeneration.

With the marked evidence of peripheral vascular disease elsewhere in the body and the lack of sclerotic changes in the retinal

vessels, it was felt that these macular changes are probably the result of angiospasm and represent the end-stage of central angiospastic retinopathy.

Discussion. Dr. Daniel Snydacker:

The paper on central angiospastic retinitis to which Dr. Hagens referred was presented before this society in 1938 by Gifford and Marquardt. They reviewed the literature of similar conditions reported under different names—central serous retinopathy, central serous retinitis, central retinal detachment, and flat detachment of the macula. After reporting eight or 10 cases of their own, they advocated the use of the term "central angiospastic retinopathy."

I do not believe the condition they described is similar to that seen in this patient. They described the condition as occurring characteristically in young people in the second and third decades. The ophthalmoscopic findings were always minimal. Visual reduction was from normal to 20/50, 20/60, 20/70; occasionally to 20/200. There was a transient hypermetropia and, with the proper lens, vision could be improved somewhat, although generally not to normal. The attacks lasted for a variable period, usually a matter of weeks, occasionally a month or more, and then disappeared completely leaving perfectly normal vision and usually no ophthalmoscopic change, or occasionally a few yellow dots in the macular area. The condition has a tendency to recur and sometimes, after many recurrences, central vision is permanently affected.

It was emphasized in that article that it is necessary to prove a peripheral vasospasm, and that diagnosis could not be made on the eye findings alone. If angiospastic retinopathy is to be broadened to include many other related conditions, this patient might fit into that category. His condition undoubtedly has a vascular basis.

It might be pointed out that the history may not be entirely reliable, because the vision may already have been poor in one eye and the vision failed, not in both eyes at the same time, but in only the eye which until that time had good vision.

There are certain features that suggest a Kuhnt-Junius disease, but it is not absolutely typical. It would seem that senile macular degeneration could be considered, since it is now known that this condition follows a more or less hereditary pattern, each familial group being more or less similar. It might be interesting to investigate this patient's family background.

Bilateral retinal cyst

Dr. W. Devereaux Jarratt presented a white woman, aged 58 years, who had come to the clinic in the spring of 1952 because of vertigo and dizziness. It was found that she had hypertensive arteriosclerotic cardiovascular disease, with blood pressure that varied from 180/100 to 230/120 mm. Hg. With these exceptions, the physical findings were essentially negative, as were laboratory studies. She had no complaints in so far as vision was concerned, which was 20/600 R.E., and 20/200, L.E. Many years previously she had worn glasses but did not have them replaced when lost.

External examination of the eyes was entirely negative except for a near point of conversion of 120 mm. A correction of R.E., +4.0D. sph. ⊃ +1.0D. cyl. ax. 105°; L.E., +3.75D. sph. ⊃ +1.75D. cyl. ax. 105° gave 20/40+2 in each eye. An add of +2.5D. to both eyes gave near vision which allowed J3 to be read at 16 inches.

On funduscopic examination there was immediately evident a sheathing of the arterioles with arteriovenous ratio of 1:4 with well-advanced nicking. The right superior temporal and the left inferior temporal arterioles showed marked sclerotic changes; in some areas no blood seemed to be carried by the arterial tree.

As these branches were traced temporally to the mid- and far periphery, a smooth bullous detachment was encountered that showed no retinal folds and did not change

in appearance with alteration of the patient's postion. The retinal vessels were markedly narrowed immediately after they left the normal retina and progressed over the sharply outlined egg-shaped eminences. The veins over the detachments were purplish in hue, whereas the arterioles were but white, thickened, obliterated channels with no evidence of blood in them.

Focusing deeper through the inner walls of the detachments, a snowflake inner area was reached beyond which vision was limited. The detachment in the right eye had a more superior position than that in the left but they were the same in size.

Peripheral fields through constricted pupils showed no evidence of change; however, on dilation of the pupils and with the eye abducted, a nasal loss of field, sharp with steep limits, was noted.

The patient has been seen at weekly intervals. No appreciable change in the findings has occurred except that the vision has been improved with correction to 20/30 + 2 in both eyes.

Discussion. Dr. Stefan Van Wien:

Dr. Jarratt is to be congratulated for an excellent clinical presentation of this rare anomaly which, in the absence of subjective complaints, could be easily overlooked on routine ophthalmoscopic examination, especially if the direct method only were used.

Weve emphasized the clinical importance of differentiation between cystoid degeneration of the retina and true cyst formation. In cystic degeneration there is primary disintegration of neural elements followed by filling of a vacuum with fluid. In true cyst formation, fluid formed between layers of the retina actively progresses to separate these layers. Cystic degeneration is seen in the periphery of the retina as well as in the macula.

Five types of cyst formation in the retina have been observed by Duke-Elder. The first group consists of cysts that are congenital in nature, which occur in microphthalmus as well as in otherwise normal eyes; in the latter case they are often bilateral and symmetrically situated in the inferior temporal quadrants. The second group is the terminal stage of cystic degeneration. In the third group, cysts form in an atrophic area of the retina after trauma or choroiditis. In a fourth group, fibrous bands of a shrinking vitreous cause cyst formation. Finally, certain parasites will cause cyst formation.

The case shown here belongs to the first group. It is generally considered to be a congenital anomaly. The cyst forms in the inner or outer nuclear layer of the retina, rarely in the ganglion-cell layer.

The ophthalmoscopic picture is characteristic. There is a sharply outlined bullous detachment that does not change position. No moving folds are observed. Breaks in the inner wall are seen that do not lead to detachment. The very thin inner wall of the cyst contains the retinal vessels which may be greatly attenuated or completely obliterated. The shiny reflexes mentioned are an optic phenomenon due to the reflected light of the ophthalmoscope. Visual fields show steep borders.

Weve holds that peripheral cystic degeneration of the retina is rarely the cause of detachment. This is corroborated by Adalbert Fuchs, who found breaks in the retina behind areas of Blessig-Iwanov degeneration. On the other hand, a break in the outer wall of a retinal cyst will cause retinal detachment, and it is quite possible that frequently cases of so-called idiopathic disinsertion of the retina actually are due to cysts. Cases have been reported where a break in the inner wall of the cyst was followed by spontaneous reattachment.

There is a difference of opinion as to the treatment of retinal cysts. Weve and Stallard are in favor of early diathermy treatment; Schepens and others follow a conservative course. Visual fields should be checked every few months for evidence of progress. Also, the appearance of moving folds or breaks in the outer wall would be an indication for active interference. The

surgical results reported have been uniformly favorable.

Bowen's disease

Dr. Robert Willard presented a 60-year-old white woman, born in Poland, who has lived in this country for 43 years. Her general health is good except for chronic cholecystitis. She was first seen in November, 1951, with complaint of an irritated, red, tearing right eye for the past year and a half.

Vision with correction was 20/25 in each eye. Examination of the left eye was essentially negative except for incipient senile cataract.

Examination of the right eye revealed mild edema and hyperemia of the upper lid with ulceration and verrucous growth on the outer one third of the lid border; marked hyperemic conjunctiva of the upper lid and superior portion of the globe, which bled readily on manipulation; a scant stringy mucus secretion; a superficial flat pannus extending onto the cornea above Bowman's membrane for a distance of three mm. in the 10- to 3-o'clock area. The intraocular pressure was 23 mm. Hg (Schiøtz). Incipient senile cataract was present. Fundus examination was normal.

A working diagnosis of acute trachoma was made. Scrapings and cultures of the conjunctiva revealed hemolytic Staphylococcus aureus. No inclusion bodies were found on repeated scrapings and arduous microscopic study. Over the next two months systemic aureomycin and Gantrisin were tried unsuccessfully; 30-percent sodium sulfacetamine solution used locally cleared the secondary infection and gave some symptomatic relief.

Cultures for fungi and higher bacteria were negative. A biopsy of the superior palpebral conjunctiva revealed only chronic inflammation with round-cell infiltration. The pannus continued to progress and a malignant growth of the conjunctiva was suspected.

Contact with the patient was lost for one month and, when she returned, there was a significant change in the appearance of the right eye. The pannus was now elevated, especially in the 10- to 3-o'clock areas, and had a gray gelatinous appearance.

Dr. Derrick Vail suggested the diagnosis of Bowen's disease. A biopsy of the pannus was taken and microscopic examination by Dr. Bertha Klien confirmed the diagnosis and revealed no infiltration through the basal-cell layer of the epithelium.

The remainder of the pannus was surgically excised and a peritomy was done in the 9- to 3-o'clock area. Because of the extensive involvement of the bulbar and palpebral conjunctiva total excision would have meant enucleation of the eye and excision of the upper lid. A more conservative method of treatment was chosen because of the precancerous nature of the disease.

X-ray therapy was selected rather than Beta radiation, due to the fact that no applicator could well reach into the superior culde-sac.

Through the upper lid, with a half-moon cone at a distance of 4.5 cm., a total of 4,200 r was given in fractionated doses through standard filters over a four-week period. About six weeks after the last treatment, the patient had a violent irradiation reaction which lasted about five weeks and gradually subsided.

There is now some residual lid edema and erythema from irradiation, no recurrence of the pannus, slight hyperemia of the superior bulbar and palpebral conjunctiva, partial loss of the cilia which are starting to regrow, mucus discharge, and increased anterior cortical and posterior subcapsular changes in the lens but none, as yet, typical of irradiation cataract.

Intraepithelial epithelioma occurs in older age groups—60 years and older. It is usually found in males and is most commonly unilateral. It appears on the cornea or conjunctiva as a diffuse, slightly elevated vascularized gelatinous or granulomatous-looking le-

sion. Its origin is in the epithelium and it may be confined there for a long period without showing infiltrative tendencies. The inflammatory reaction which accompanies the lesion may mask its tumorous nature (as in this case).

Bowen, an American dermatologist, in 1912 described two patients with a chronic skin lesion which he regarded as belonging to a group of precancerous dyskeratoses. Although subsequently described many times in dermatologic literature, it was not until 1942 that it was reported in the eye by McGavic, who described five cases.

Since then only six or eight cases of ocular Bowen's disease have been reported. Reese, however, states that it is much more common than is suspected, and has 18 recorded cases at the Institute of Ophthalmology of Presbyterian Hospital in New York. Several cases are recorded also at the Cleveland Clinic.

The classification of the disease has been a controversial subject. Some classify it as a form of squamous-cell carcinoma in situ, others as a precancerous lesion with malignant possibilities such as Paget's disease, and so forth. Malignant change occurs in 40 percent of mucous-membrane lesions and three percent of skin lesions.

The disease should be differentiated from Mooren's ulcer, filtering cicatrix of the cornea, corneal pannus from any cause, fatty degeneration of the cornea, epithelial dystrophy, and pterygium.

Pathologically, the epithelial hyperplasia is entirely intraepithelial. The basal cells proliferate and lose their palisaded arrangement but remain intact as a layer. Poikolokarynosis is the term used to describe the variations in size, shape, and staining characteristics and loss of cell polarity. So-called monster cells and "corps ronds of Darier," both large cells, are present. Mitotic figures are common. Keratin pearls may be present. Beneath the epithelium, lymphocytes and plasma cells are present.

The preferred treatment of intraepithelial epithelioma is complete surgical excision. Biopsy of the lesion is not contraindicated. Irradiation is not satisfactory because the tumor is not very sensitive. Due to its precancerous nature, however, temporizing is less dangerous than with a true basal or squamous-cell carcinoma.

Scientific program

Effect of cigarette smoking on flicker fusion thresholds

Dr. Lawrence L. Garner, Dr. E. Franklin Carl, and Dr. Erwin E. Grossmann (by invitation) discussed the "Clinical observations of the effect of cigarette smoking on the flicker fusion threshold of normal smokers and nonsmokers."

The effect of cigarette smoking on the flicker fusion threshold was observed in 55 smokers and 53 nonsmokers. Of this group 106 were women—96 student nurses in the age group 18 to 23 years and 12 graduate nurses ranging in age from 24 to 53 years. The two men were residents in ophthalmology.

Thirty-five of the subjects revealed an increase in the flicker fusion threshold following smoking, 21 revealed a decrease, 40 presented no alterations, five showed an initial rise followed by a secondary fall, and seven could not be considered good candidates because of erratic results.

From their study, the authors are of the opinion that nicotine initially affects the central nervous system and that this effect becomes masked by the subsequent effect on the blood vessels. The variability in response may be based on individual sensitivity to nicotine and individual variations in the reactions of the vascular system to nicotine. Decrease in flicker fusion frequency could be avoided or almost completely avoided by substitution of a denicotinized cigarette for an ordinary cigarette, suggesting that nicotine is the cause for decrease in flicker fusion frequency. The measure of this response may become a prac-

tical means of detecting hyperreactors to nicotine.

Discussion. Dr. Andrew C. Ivy:

I should like to introduce my discussion by citing two physiologic precepts:

First, I have told my students many times that when we learn how to measure carefully a basic physiologic function or activity, sooner or later we find a practical application for the measurement in either diagnosis or therapy, frequently both. I point out that precept to emphasize what has happened in medical history and especially to stimulate interest in basic physiologic phenomena for which at present we do not have an immediate practical application.

The second precept is that physiologic changes unquestionably occur prior to morphologic changes, and that is particularly true of gross morphologic changes.

There is nothing particularly new about either of these precepts but both apply to this attempt to find out if the measurement of the critical frequency of fusion of light impulses might be of some diagnostic or therapeutic value.

The idea that a spasm or hypertonus of the musculature of the blood vessels of the retina may precede the semigross to gross changes seen with the slitlamp or the ophthalmoscope is not new. This was pointed out some 25 or 30 years ago by Duke-Elder and more recently by Dr. Gifford. This general idea is applicable to changes which occur in the peripheral vessels in hypertension, particularly essential hypertension.

Early in the disease there is no morphologic evidence of disturbance in the histology of the arterioles. But after hypertension has been established for one or more years, depending upon how benign it is, morphologic changes are found. The physiologic concept is that there is either excessive vasomotor nervous activity upon the peripheral vessels or some substance of the nature of a hormone or a hypertensive substance acting upon the peripheral blood vessels. That increases the viscosity of the myoplasm of the smooth muscle cells which interferes with the vascularity of the blood vessel wall and, in the course of time, the morphologic changes can be observed with the microscope and, later on, with the unaided eye. I picture such changes as occurring in the retinal blood vessels.

When Dr. Krasno and I started to apply the measurements of the flicker fusion threshold in the attempt to measure some clinical symptoms, we picked out the subjective sensation of fatigue. We would like to have found a way to measure objectively the subjective report of the patient who states that he feels tired. We found that the correlation between the feeling of tiredness and the flicker fusion threshold under controlled conditions was only about 0.7, or 70 percent.

We then looked into the question of the relation of a light meal or a heavy meal, time after meals, smoking, climbing stairs, and the tension caused by an examination to the reaction on the threshold. We found in these preliminary studies essentially what Dr. Garner and his colleagues have reported. We gained the impression in the case of smokers who feel a "let down" because they have not had a smoke for awhile that, if they smoked and inhaled, their flicker fusion threshold would increase and they would have a so-called "pickup." We found also subjects in whom the flicker fusion frequency was depressed, indicating hypothetically an anoxia of the retina or visual mechanism.

We studied the environmental factors which affect the flicker fusion threshold when we started to use it as one of the psychomotor measurements applied to persons subjected to the anoxia of low pressure or high altitude in a pressure chamber. Measurements of the flicker fusion threshold and the brightness discrimination were the most sensitive tests, other than the determination of the oxygen saturation of the blood, by which to detect the development of anoxia as it appears on decreasing the pressure in

the pressure chamber. We now know that visual functions which can be measured very accurately are sensitive indicators of anoxia.

After the war, Dr. Krasno tried to determine whether there might be any relation between changes in the vascularity of the retina and the flicker fusion threshold. It did not take long to find out that such a relation existed, but he realized that the normal range of activity of the flicker fusion threshold varied in different individuals throughout the day.

In order to use the test effectively, he used the patient's threshold at the time the measurment was made and then, to test the presence of spasm or hypertonus, he chose nitroglycerine because of its rapid vasodilator action.

Dr. Puntenney has shown that it takes rather large doses of nitroglycerine to show a measurable change in the size of the blood vessels when viewed with the slitlamp, and then it occurs only in a few individuals. But it was thought that the physiologic change detected by the flicker fusion threshold might be more sensitive than the measurement of blood vessels made with the slitlamp.

The variations observed by Dr. Garner and his colleagues are, I believe, to be expected, especially when one recalls the work which has been done on the effect of smoking on skin temperature, which reflects the blood flow through the skin. In some 80 to 90 percent of individuals, if the smoke is inhaled, the skin temperature is decreased; in from 30 to 40 percent of individuals it is decreased two degrees or more. In a much smaller percentage, between four and five percent, the so-called hyperactive group, the skin temperature will be reduced from eight to 12 degrees.

The study of Gifford and Marquardt, made about 15 years ago, showed that the decrease in skin temperature was marked in patients with tobacco amblyopia and with what was diagnosed as tobacco angiospastic retinitis. They thought that the marked changes in the skin temperature and the retina might, in the latter group, be ascribed to an allergy to tobacco.

Although I am willing to accept as rational, from the viewpoint of physiology, the explanation of Dr. Fabricat, we will not approach a true explanation of these variations until we study not only the flicker fusion threshold but the skin temperature and pulse response to smoking. The results of such a correlated study would probably illuminate the mechanism of the cause of the variations observed in the response of different individuals.

There are retinal functions with which ophthalmologists are familiar, which can be measured with a high degree of accuracy and which, if studied, will prove in the course of time to be of value in diagnosis and therapy. I believe that in another five or 10 years the testing of the flicker fusion threshold will be recognized as a standard test for certain specific purposes.

There is one important observation which pertains to the general subject of the duration of the effect of nicotine on individuals who are hyperreactive. I shall cite two cases:

A patient who smoked about a pound and a half of tobacco a week for 20 years developed symptoms of intermittent claudication and was advised to stop smoking. Four months passed before all the symptoms cleared.

The second patient was a cigar smoker who had precardiac symptoms resembling those of angina pectoris. He showed a coronary constriction on the basis of the flicker fusion threshold response to nitroglycerine and was advised to stop smoking. Four weeks passed before the flicker fusion threshold response to nitroglycerine became normal.

If any ophthalmic condition suspected as due to tobacco is present, you will not necessarily obtain the correct answer simply by having your patient stop smoking for a few days. He may have to stop smoking for weeks or months before the condition reverses to normal. And no change will occur

if an irreversible change in the physiologic mechanism has taken place. In the presence of a drug or poison which has a cumulative effect, one cannot expect abolition of the effect to occur in a few hours or days by simply withdrawing the drug or poison.

Dr. Harold M. Brill:

The flicker fusion threshold test is not new, and the physiologic concepts behind the test are rather universally accepted by scientists and the medical profession. You all know the work of Paul Miles of Saint Louis, who designed a flicker photometer on the principle of a stroboscope and used it in mapping out the visual fields. Berg, in his monograph in the *Acta Psychiatrica et Neurologia*, has utilized the flicker fusion test for studies on neurosurgical patients to detect the presence or absence of brain tumors. Various internists have employed this test in detection of coronary spasm, coronary occlusion, angina, and related diseases, with various results.

Our experience has been in the field of obstetrics, where we believe the test has great potential value as a diagnostic aid in the preclinical determination of toxemia of pregnancy. It is an accepted fact that vascular hypertonus brought about by spasm is an integral part of this disease entity. We feel that the flicker fusion test would indicate to us in any given patient whether or not she will develop any clinical signs of toxemia of pregnancy, and have been able to detect accurately the advent of the disease from six weeks to three months prior to the appearance of the known clinical signs of the disease.

A recent paper in the *Journal of the American Medical Association* reports that the vascular system of certain individuals was found to be sensitive to the smoking of cigarettes, whereas that of other individuals was not appreciably affected. This being true in a group of patients with coronary disease, may also be true in the group studied by Dr. Garner and his co-workers.

Any individual, therefore, who shows a positive flicker fusion threshold after smoking is one who is sensitive to the effect of tobacco, and cardiovascular patients who show a positive flicker fusion threshold response should not be allowed to smoke because of further harmful effects to the vascular tree. Dr. Garner has thus inadvertantly proven that the performance of the flicker fusion test should become a definite part of the complete cardiovascular study in any individual. We have prohibited smoking for from 12 to 24 hours before the test was performed so that the effect of smoking would not confuse our results.

In Dr. Garner's study, the postsmoking flicker fusion threshold rates were averaged after taking from six to 10 readings immediately following. The question arises as to whether or not too many readings were taken in too short a time, thereby allowing the fatigue factor to enter into the results. His readings were taken at various intervals from three to 15 minutes, postsmoking.

We took only three readings following the administration of nitroglycerine. It is significant that the denicotinized cigarette produced no change in the flicker fusion threshold. Moreover, patients placed in a smoke-filled room showed the same effect on the flicker fusion threshold as if they had actually smoked cigarettes. Again, nitroglycerine postsmoking returned the flicker fusion threshold to normal and, in most instances, reduced the recovery time to somewhat less than one half.

Dr. Garner stated that funduscopic examinations of these patients at no time revealed evidence of vascular spasm. I should like to ask whether he believes the cigarette effect on the flicker fusion threshold to be of central or peripheral origin. Also, does he believe the picture of the vascular tree as seen in the ocular fundus can be considered as a mirror, or as part and parcel of the general picture of the entire vascular tree of the individuals?

His subjects were all young, with no preexisting vascular changes. Our patients also

would include a similar age group; that is, those in the child-bearing age. In two of his cases, the subjects were older, and the effect on the vascular tree is not clearly understood.

The paper makes the important point that nicotine initially affects the nervous system, then the effect is masked by the blood-vessel effect. Undoubtedly, the flicker fusion threshold reaction is valuable for the detection of sensitivity to nicotine.

We would like to make a plea for further investigation in all fields of medicine, to determine whether this easily conducted and economical test is applicable in other branches, and thus give additional diagnostic data. Only by thorough and complete investigations of this type can we arrive at the final evaluation of the flicker fusion threshold test.

Dr. Noah D. Fabricant:

In reviewing medical journals from all over the world I have been impressed by the fact that there has been very meager investigation on the effect of cigarette smoking on human beings in health and disease. I am also impressed with the great numbers of advertisements of cigarettes that appear in medical periodicals, especially American.

Smoking is not a moral issue. However, we physicians know little about the effects of smoking because we have paid little attention to the problem. Dr. Garner's work is not only a step in the right direction but in a decisive direction, since his work may appeal to other investigators in the field of ophthalmology.

He has pointed out that flicker fusion may indicate excitability or depression of the visual system, perhaps of the entire central nervous system. He also makes the extremely important statement that "flicker fusion has no correlation with visual acuity; it is not an index of visual acuity." If it were considered to be an indication of visual acuity we could expect a deluge of claims by cigarette manufacturers to the effect that smoking their particular brand would aid vision. I am pleased, therefore, that he defined the limitations of the flicker fusion threshold.

It has been pointed out that the flicker fusion threshold is related to the vascular supply of the retina or the brain, and that the flicker fusion threshold increases when there is increased blood supply, and decreases when the blood supply is diminished.

All but two of Dr. Garner's subjects were females. I should be interested in knowing whether similar results could be obtained with a comparable group of males.

In my own study, I limited myself to 50 presumably normal males. The flicker fusion test was found to be increased in 40 and decreased in 10 subjects. I do not know whether that proportion would hold true for a larger group.

Again, it has been shown that, theoretically, vasoconstriction should produce a decrease in flicker fusion threshold if the blood supply to the visual pathways is impaired. Unfortunately, the various hypotheses advanced, including my own, do not adequately explain the increase in flicker fusion threshold which occurs after smoking.

There is substantial evidence that nicotine may cause an increase in flicker fusion threshold; also, this appears to be avoided by the use of denicotinized cigarettes. My impression is that Dr. Garner and his associates intend to carry their study further, and I might suggest that flicker fusion threshold studies be made after smoking and then after drinking a cocktail, to see whether one may smoke to cause vasoconstriction of the small blood vessels, and then take an alcoholic drink to overcome it by producing vasodilation. I would also like to suggest the use of inhaling straws; the use of the various types of filters. There is one filter on the market which is so effective that one might as well inhale hot air. I would also like to know the effect of smoking corn silk. We know that it does not cause cutaneous temperature drops in the fingers.

I was interested in Dr. Garner's comment

on the effect of noise on the readings. Some years ago I did an extensive study on the pH of the nasal secretions in situ, and found the influence of noise caused a transient variation from a normal nasal acidosis to an alkalosis which lasted for a few minutes. However, as I continued my study I finally decided that this was due to hyperventilation. Perhaps noise causes subjects to hyperventilate. Perhaps it would be possible to have people hyperventilate and then do flicker fusion threshold readings on them.

I think Dr. Garner's work is important, especially if future studies will demonstrate that this test may be used as a practical means of detecting hyperreactors to nicotine.

Dr. Lawrence L. Garner (closing):

I should like to thank Dr. Hughes for his help and suggestions during the course of this project which began in 1950. We were all confused at the start because of the variables, variations, and the peculiar results found upon repeat tests in the same subject. As the work progressed our findings began to take a pattern indicating that all subjects did not react alike and, based upon this, our classification resulted.

There are questions that we are unable to answer at this time, but it is our belief that nicotine initially affects the central nervous system, and that this effect becomes masked by its subsequent effect on the blood vessels, the combination producing the results noted, and the latter being dependent upon how the individual reacts to nicotine. This difference in reaction probably accounts for the various results noted.

We cannot explain why 40 of our subjects presented no change after smoking ordinary cigarettes, unless we assume that their response to nicotine was negligible or that the test was inadequate.

As to the effects on the vascular tree of the retina, I believe it was Cusick and Herrell who reported observations of the retinal vessels following smoking. They employed a modified Morgan graticule in a Keeler ophthalmoscope and were thus able to project fine lines onto the retina. By this means they were able to detect about a 20-percent decrease in the caliber of the vessels near the disc, but of temporary duration. It is obvious that the ordinary ophthalmoscope would reveal nothing or practically nothing.

Buried orbital implant

Dr. Maurice D. Pearlman spoke on "A buried orbital implant of plastic sponge."

A new soft, biologically inert, plastic sponge, having certain theoretical and practical advantages was used for orbital implants in 69 patients, ranging in age from four to 77 years. Observations extending over 24 months indicate that the material is well tolerated and the cosmetic results are satisfactory. There is a minimum of serious complications and relative freedom from hygienic problems. About nine percent of the cases were unsuccessful because irreparable exposures of the implant and consequent infection developed during the early postoperative weeks. In the remaining 91 percent, the implant has remained in place for as long as 24 months.

Richard C. Gamble,
Recording Secretary.

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THE SCLERAL RESECTION OPERATION

The modern era of retinal detachment surgery dates back to 1919 when Gonin presented his thesis of curing the detachment by cauterizing the hole. The present methods of diathermy operation are successful in the majority of cases. Statistics vary as to the selection of cases but an over-all cure of 70 percent is expected by most ophthalmologists who do any volume of detachment surgery. In an effort to reduce the minority percentage that fail there has been a revival of interest both in this country and abroad in the scleral resection operation.

At the American Medical Association meeting in New York City, there were two presentations on the scleral resection operation. Kronfeld and Pischel reported on their results on 200 cases and Schepens in his

discussion mentioned 200 cases performed on the Retina Service of the Massachusetts Eye and Ear Infirmary. This is by far the largest series that has been reported. Though the operation was first performed in 1903, Leopold (1945) was able to collect only 121 reported cases in the literature.

Pischel and Kronfeld in their presentation and in their paper in the June issue of THE JOURNAL summarized the indications for performing the operation. To this might be added staphylomas of the sclera as reported by Vail a few years ago. Undoubtedly these recent reports will stimulate further interest and more ophthalmologists will be trying this as a primary procedure rather than as a last resort.

The majority of cases in which this operation is indicated are those in which there are so-called fixed folds. These may be meridional, equatorial, or star-shaped. They are characterized clinically by failure of the retina to settle with bedrest.

There is some difference of opinion as to whether the immobility of the retina is caused by contracture of the posterior cortical layer of the vitreous or shrinkage and atrophy of the retina. The modus operandi of the operation is also in question. One theory is that the purpose of the operation is to provide a smaller sphere whose inner surface may fit the shrunken retina. Another theory is that by invaginating the choroid, with or without a thin strip of sclera in the base of the resected area, contact is made with the retina and, as a choroidal retinal adhesion forms and flattens out, the retina may be anchored securely.

For the operation to be successful the prerequisites of any detachment surgery must be met, that is, the hole or holes must be closed with sufficient drainage of subretinal fluid to permit choroidal retinal adhesions to form. Fortunately, the success of the operation does not depend on whether the primary fault lies in shrinkage of the retina or vitreous contracture. Both factors probably play a part though the primary factor in these relatively immobile detachments is probably caused by some disturbance in the vitreous.

The operative techniques vary considerably though essentially there are but two principals involved. One is to resect the entire thickness of the sclera, the other to leave a thin layer of sclera in the base of the resected area—the so-called lamellar resection or scleral buckling. Both operations can be fraught with many difficulties and, in most instances, the loss of formed vitreous leads to disaster.

There is no question that the scleral resection operation will cure some cases of retinal detachment not cured by ordinary diathermy procedures. It is not an operation to be used indiscriminately and it does not permit the operator to be less thorough in his preoperative search for retinal breaks. The technical difficulties can be much greater than in the regular diathermy procedures.

The scleral resection operation to be most effective should be used as a primary procedure. The indications are now fairly well agreed upon. The occasional operator would probably do well to by-pass the one-eyed patient or the ones in whom the bilaterality of the detachment is but a matter of time. Like any other operative procedure it may prove relatively easy or the eye may be irreparably damaged.

Further clinical, pathologic, and experimental observations will no doubt increase our knowledge of the factors involved both in causing and in curing retinal detachments. With the increasing longevity of the population we can also expect an increase in the incidence of retinal detachments. A word of caution against the indiscriminate use of the operation would seem indicated, as well as a word of encouragement when the indications are clear cut.

P. Robb McDonald.

OBITUARIES

JOHN NORRIS EVANS

An appreciation

Reflecting on the kindly, whimsical, distinguished, and very distinctive personality, John Norris Evans, one must start with the story of a normally vigorous infant with curly red hair, not yet walking, who was stricken with poliomyelitis and left with extensive paralysis of the lower extremities.

John Norris Evans, M.D.

When convalescent, his mother, instead of carrying him about and pampering him, put him on the floor and let him struggle to get about without aid. This forced development of his remaining physical resources set the pattern for an entire life, characterized by great courage, self-reliance, and indomitable perseverance. Later the same sympathetic and loving, but not indulgent, mother would send him off on a pair of crutches telling him to "start early if you want to get to the game." He often remarked that his mother had prevented his spending his life as an invalid in a wheel chair. Following her admonition, he started early ever afterward. Eventually he developed great strength in his hands and arms and a powerful shoulder girdle. One leg could support his weight, but the other remained quite useless and rather short—he spoke of it as "just an ornament." He became a strong swimmer, learned to get along on one crutch and would entertain his playmates with a variety of stunts that required special kinds of strength and dexterity. His extracurricular activities did not prevent normal progress through grade school and Erasmus Hall High School from which he was graduated in 1912. He entered the Long Island College Hospital in the fall of the same year and received an M.D. degree in the class of 1916.

A year of internship at the Bushwick Hospital, Brooklyn, was followed by a residency in ophthalmology at The Brooklyn Eye and Ear Hospital. Dr. Evans soon established a large private practice and advanced to positions of leadership at The Brooklyn Eye and Ear Hospital, eventually becoming chief of one of its several eye services. Not content with routine clinical practice, he experimented in the fields of optics and ocular physiology. His published results earned him the Lucien Howe Prize awarded by the Medical Society of the State of New York, membership in several distinguished societies, and an international reputation. He became known as the "father of angioscotometry," a new field of clinical and investigative study of the retina in health and disease. His observations in this realm formed the subject matter for a book entitled *Clinical Scotometry*. An achievement that brought him great satisfaction was his appointment, in 1935, to the professorship of ophthalmology at his alma mater. He was forced, by ill health, to resign from that post as well as to curtail many other activities in 1948. Another honor of which he was especially proud came from his alma mater in 1951 when he was awarded the alumni prize for outstanding accomplishment.

Dr. Evans married Miss Elizabeth Adams

of Brooklyn, and they were a devoted couple. It was a great blow to him when his wife suffered a cerebral accident and died a few months later on January 4, 1953. His already poor physical condition rapidly declined until his death on April 8th, at the age of 62 years.

Dr. Evans was a fellow of the American College of Surgeons and a diplomate of the American Board of Ophthalmology. He was a member of the American, New York, and Brooklyn Ophthalmological Societies and of the American Academy of Ophthalmology and Otolaryngology. He was also a member of the Medical Society of the County of Kings, the New York State Medical Society, the American Medical Association, and the Associated Physicians of Long Island. At the time of his death he was a member of the consulting staffs of The Brooklyn Eye and Ear, The Brooklyn, the Long Island College, and St. Peter's Hospitals, and a consultant to the Ophthalmological Foundation and the National Society for the Prevention of Blindness.

Widely renowned for his scientific accomplishments and very successful as a clinician, he had an unusually large circle of admiring and devoted friends who knew him as a modest, kind, generous, and loyal companion, unaffected by professional attainments.

J. Arnold deVeer.

CHARLES BERNARD GOULDEN
(1879-1953)

Many Americans recall with gratitude the privilege of knowing Mr. Goulden during the years of his service to Moorfields, and particularly during the last war. His lectures on "Injuries to the eyes" were especially beneficial to the medical officers of the Allied Forces stationed in London and its environs.

Charles Bernard Goulden, consulting ophthalmic surgeon to the London Hospital and consulting surgeon to the Royal London Ophthalmic (Moorfields) Hospital, died at his home at Shepreth near Cambridge on September 20th at the age of 74 years.

Educated at St. Edmund's College and at Downing College, Cambridge, Mr. Goulden completed his medical training at the Middlesex Hospital. From a house appointment at Middlesex, he went to Moorfields, later served as demonstrator in anatomy at the University of Bristol, and finally located at Oldham where he was appointed to the staff of the Royal Infirmary.

Mr. Goulden left Oldham in 1916 to serve in France with Sir William Lister. Later he was put in charge of an ophthalmic center at Rouen. After the war, he was offered an appointment at the London Hospital and returned to the city. He was on the staff of the Victoria Hospital for Children in Tite Street, the Queen Alexandra Hospital, Millbank, as well as on the staff of Moorfields where he became dean of the Medical School. During the second world war, among other arduous assignments, he was adviser to the Ministry of Health on ophthalmic problems of the Emergency Medical Service.

During the years 1944 to 1946, Mr. Goulden was president of the Ophthalmological Society of the United Kingdom which office he filled with great distinction.

"All of his friends must feel deeply the passing of one whose influence on the ophthalmological world, especially on the side of education, has been so considerable and whose charming and often amusing companionship was always so acceptable." (*The Lancet.*)

XVII INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

A Message to North American Ophthalmologists

For the second time since the First International Congress of Ophthalmology was held in Brussels in 1857, the official congress will be held on the North American con-

tinent. While the congress is scheduled to be held every four years, only 16 have been held to date; the XVII congress will take place in Montreal and New York in September, 1954. The previous International Congress of Ophthalmology held in America was the Fifth, held in New York City in 1876. Interruption of the succession of congresses was the result of war which precluded the attendance of delegates from a number of the nations whose national ophthalmological societies held membership in the International Federation of Ophthalmological Societies or because it was scheduled for a time when international conventions were banned for one reason or another.

Many American and Canadian ophthalmologists have attended International Congresses in other countries and have built up a warm personal regard for their colleagues in other lands. Many of us have repeatedly visited clinics and hospitals in foreign cities and have studied under the able faculties of European institutions and private instructors. There has developed in the United States and Canada a strong desire to be hosts to the next International Congress and the decision of the International Council to include Canada in the XVII International Congress met with favor throughout the world.

Ophthalmologists of the United States and Canada have everything in common that can possibly be shared by two nations. A reciprocal certification of specialists by the respective national specialty Boards of Ophthalmology and Otolaryngology is based on mutual respect of the integrity of the individuals and of the educational and training requirements and achievements of the two countries. As joint hosts to the XVII International Congress we extend a mutual welcome, a common hospitality, and a single program.

While the extension of the congress to Montreal makes the XVII the longest of any session, the record number of papers, motion pictures, exhibits, and anticipated attendance fully justify the two-day extension of the official meetings.

The congress deserves and will exemplify the united support of all North Americans. Late changes in plans have shortened the time of notification of the place and character of the meeting and, incidentally, added to the expense of the organizing committee. Your opportunity to take part in this congress is unrestricted, your complete support solicited. Send your financial contribution and your advance registration at the earliest possible moment. All contributions will be personally acknowledged. Make your plans now for September, 1954. Bear in mind that the Academy meets the following week in the same place.

William L. Benedict,
Secretary-General.

CORRESPONDENCE

Blind and Blindness

Editor,
American Journal of Ophthalmology:

May I congratulate you on the excellence of the paper, "Blind and Blindness," by Mrs. Thelma Charen (36:839 [June] 1953)? There can be no greater stimulation than that derived from the probing into the background of such seemingly common concepts among different nations. Etymology can become a valuable aid to the practicing physician by disclosing hidden meanings which will open new vistas in physiology, pathology, and therapy.

The author of the paper shows an astonishing knowledge of the unexpectedly wide range of her problem with its various derivations. Yet she has omitted two concepts that should be added. I am speaking of ***amaurosis*** and ***amblyopia***. Both words are of Greek origin and are to be found in the writings of the classical authors, and the first has played an important part in ancient Greece's medicine.

In Hippocrates, ἀμαύρωσις means obscuration and weakening of vision, but, interestingly enough, Aristotle, in his famous work "On Soul" uses the word to indicate a weakening and obscuration of the intelligence and of the whole mind. The adjective ἀμαυρός means dark, obscure, and thus this aspect of blindness is stressed by the use of ἀμαυρός instead of the common τυφλός, which has been so ably analyzed by Mrs. Charen.

By the way (and perhaps this fact has prompted the author's omission), up to this day, classical philologists have tried in vain to detect the etymologic root of amaurosis—it has remained ἀμαυρόν to them, thus preconising the somewhat mystical development of this expression.

For today, amaurosis has entered the large realm of psychology or at least psychosomatic medicine. Though some physicians still use it for expressing "common" blindness, it has more and more come to mean total or partial loss of sight without apparent organic defect. In psychiatry, we find amaurosis connected with idiocy in certain cases of hereditary feeblemindedness.

Amblyopia (ἀμβλυωπία), a concept with a perfectly clear etymology, was also used by all great ancient Greek writers. It means feebleness or weakness of vision, and we encounter it in Hippocrates' aphorisms. Plato uses it in his dialogue, "Hippias Minor," and Plutarchus in his "Ethics," whereas in Aristotle we only find the adjective ἀμβλωπός.

Apparently, amblyopia has much more rarely been used in a figurative and metaphoric sense than has amaurosis. It has, so to speak, remained in the realm of medicine proper and even resisted the temptation to invade modern psychiatry including depth psychology which is more and more inclined to usurp amaurosis for the meaning of hysterical blindness.

(Signed) Henry W. Brann, Ph. D.
Research Psychologist
Washington, D.C.

BOOK REVIEWS

ACCIDENTES OCULARIS DEL TRABAJO. By Jose A. Sená. Buenos Aires, Argentina, El Ateneo. 133 pages. Price: Not listed.

This second edition of a monograph on industrial eye injuries, first published in 1941, and the recipient of the Lagleyze prize of the Medical School of the National University of Buenos Aires, is the fruit of many years of labor and thought by the chief of the Eye Section of the Argentine Ministry of Public Health (previously the Department of Hygiene). The author has compressed into only 133 pages a complete review of the subject, including an excellent summary of the mechanism and pathology of eye injuries and occupational eye diseases.

The main purpose of the monograph, however, has been to help in the standardization of the appraisal of loss of visual functions from eye injuries. Argentine legislators in establishing workmen's compensation, which dates from 1915, failed, as other legislators have done, to define many items in the law. Industrial blindness is not defined, nor is a schedule given for partial loss of visual functions, and the role of the fellow eye in the appraisal is only vaguely dealt with in the law.

As elsewhere, it devolves upon ophthalmologists, who are called upon to act as technical experts to the lay adjudication officers, to supplement this omission. This has been the task of the author, and his proposed table of compensation for eye injuries received a prize at the first Argentine Congress of Industrial Hygiene in 1950, and is apparently gaining acceptance outside of the Argentine in neighboring countries. It is in line with, and not much at variance from, the tables recommended in 1941 by the Committee on Visual Economics of the American Medical Association. The special features are:

Compensation is based on percentage loss of both eyes, with different tables used for binocular injuries and for defects of the fellow eye antedating an eye injury.

Compensation for monocular and binocular aphakia is for corrected visual acuity, di-

vided by two. (Evaluating monocular aphakia as the loss of an eye, the author argues, destroys the incentive of the insurance carrier to provide for extraction, with the best possible results and with benefit to the worker.)

Compensation is based on corrected visual acuity, except where the refraction error is the result of the injury.

As in all newly industrialized countries, industrial accidents in the Argentine run high and constitute the bulk of eye injuries. The author therefore makes a plea for safety measures in industry, for the eye examination of all head-injury cases, however, minimal, and for pre-employment eye examination, provided it is accompanied by periodic eye examinations and conducted by a government agency.

In the author's experience, the amblyopias constitute the most difficult problem in differential diagnosis. The incidence of malingering has decreased in the course of the years, and is attributed by him to the experience gained in diagnosis and in the handling of workmen's compensation cases. Both observations, to the reviewer, constitute the most valid arguments for the use of a staff ophthalmologist attached to the administration of workmen's compensation.

Morris Davidson.

BULLETIN OF THE GREEK OPHTHALMOLOGICAL SOCIETY. Athens, Greece. Volume 19, Part 3, 1952; Volume 20, Part 1, 1952.

In Part 3 of Volume 19 the first meeting of the newly founded Ophthalmological Society of Northern Greece, which met in joint session at Salonika on September 28 and 29, 1951, with the Greek Ophthalmological Society, is reported. Of the 60-odd papers and case reports published in this issue of the *Bulletin* only a few can be singled out for special mention.

A. Trantas, who is known to ophthalmologists everywhere as the discoverer of velanoskiascopy, tells how this subjective method of determining the refractive error may be still further simplified by the use of a single movable light streak, which provides a very sensitive test for uncovering small amounts of astigmatism that otherwise might escape detection.

Prof. T. Tjanides presents his views on the surgical treatment of chronic simple glaucoma. Because of the high incidence of low-grade iritis, synechias, and cataract after any kind of fistulizing operation he much prefers Weekers' iridencleisis and nonperforating diathermy to the ciliary body. These operations are not complicated by cataract or any inflammation of the anterior segment.

K. Zervakakos reports a case of bilateral keratoconus in a patient with Addison's disease, and reasons therefrom that the etiology of keratoconus is to be sought in an insufficiency of the suprarenal glands. A decrease in sympathetic tonus is thought to result in a lowering of the pressure in the circumcorneal capillary network, with consequent malnutrition and weakening of the central part of the cornea.

I. Fronimopoulos gives a preliminary account of his experiments with injections of air under the conjunctiva or behind the globe. He found that a subconjunctival injection of air was a very effective form of treatment for subconjunctival hemorrhages, causing complete absorption within a very short time. Retrobulbar injections of air were likewise found beneficial in cases of retinal hemorrhages, but further study and observation are required in this regard. Air injections, by accelerating the processes of absorption, even though without effect on the etiologic factors, may be of great value in forestalling irreversible secondary changes.

T. Dimitriou reports on his treatment of retinitis pigmentosa with local instillation of extract of the middle and posterior lobes of the pituitary gland. Rapid subjective improvement of short duration was noted in nine out of 16 cases. There were no undesirable side effects.

A. Tranou-Sfalankakou and P. Velissaropoulos report a case of maxillofacial dysostosis in a girl who had dermoid tumors of the conjunctiva of each eye. The presence

of the latter has not been previously reported in this disease. Prof. G. Cosmetatos summarizes all the ophthalmic dissertations that have been presented since 1928 by candidates for the doctor's degree and for teaching fellowships.

Part 1 of Volume 20 is a comprehensive treatise on tuberculosis of the eye, written by I. Fronimopoulos and T. Ioannides to provide the medical fraternity of Greece with an up-to-date practical summary of what is known of the pathogenesis, diagnosis, clinical forms, and treatment of the ocular manifestations of this disease which is so common throughout Greece. The latest forms of treatment are amply set forth and critically evaluated on the basis of the authors' wide experience. There is a wealth of material in this excellently printed and well-illustrated monograph, but unfortunately, being written in Greek and not furnished with a summary in any of the more widely known languages, it will hardly find readers beyond the small circle for which it is intended.

Harry K. Messenger.

LONG-TERM DIABETES. The clinical picture of diabetes mellitus of 15-25 years' duration with a follow-up of a regional series of cases. By Knud Lundbaek, M.D., Lecturer in Internal Medicine, Aarhus University Medical School, in collaboration with Viggo A. Jensen, M.D., Professor of Ophthalmology, Aarhus University Medical School. Copenhagen, Ejnar Munksgaard, and London and New York, Lange, Maxwell and Springer, Ltd., 1953. 197 pages, supplementary tables.

This extensive survey of long-term diabetics, comprising all the known diabetics of at least 15 years' duration in the municipality of Aarhus, Denmark (some 234 in number) is of interest not only for the statistical analysis of these cases but for the exhaustive exposition of the current literature on the subject. To the ophthalmologist who, since insulin has prolonged the life of the diabetic patient, is seeing an increasing number of the serious ocular complications of the disease, the book should prove illuminating.

Of particular interest is the extensive discussion of the Kimmelstiel-Wilson syndrome. Retinopathy was present in 92 percent of the patients with renal disease and 76 percent of those without renal disease. The authors believe that it is not always possible to distinguish ophthalmoscopically between the microaneurysms and minute hemorrhages seen in the retina and prefer to classify them together as "sanguinolent spots." Exudates, venous changes (dark, congested, broad veins), proliferative changes, and pigmentopathy of the macula are believed to be the other important retinal changes. The pigment changes in the macula were found rather frequently in this series although they are not commonly recognized as a part of the diabetic state and must be differentiated from senile macular degeneration in the older age group and heredodegenerative changes in the younger patients. True arteriosclerosis is not regarded as essential in the production of the retinopathy although it is frequently present. Deducting the cases which showed only pigmentopathy of the macula or phlebopathy, the total series of long-term diabetics showed hemorrhages and exudates in 39 percent, hemorrhages without exudates in 28 percent, and exudates alone in four percent; eight percent of the cases with retinopathy showed proliferative changes. Changes in the veins and macula were noted especially in the young long-term diabetics. Lipemia retinalis and rubeosis iridis diabetica were not encountered in this series.

Controlled experiments with rutin and vitamin preparations did not reveal any beneficial effect from their use as determined by periodic study of the fundi.

This interesting and important analysis of the long-term diabetic is an important contribution to the literature of the subject. Ably translated by Axel Anderson, it makes fascinating reading to anyone interested in the complications found in the patient with a diabetes of long duration.

William A. Mann.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

2

GENERAL PATHOLOGY, BACTERIOLOGY, IMMUNOLOGY

Bocci, G. **Study of ocular active and passive anaphylaxis.** Boll. d'ocul. 32:237-241, April, 1953.

Fifty units of diphtheria anatoxine were instilled daily into the conjunctiva of eight rabbits and 1,200 units of antidiphtheria serum into the conjunctiva of four more rabbits. Two weeks later blood was withdrawn and, after clotting, the Praus-nitz-Kustner test was performed which gave negative results. Two animals of each group received different treatment: a) instillation of diphtheria anatoxine into both eyes—this produced a mild congestion in the conjunctiva of the previously sensitized eye; b) antidiphtheria serum instilled into both eyes produced no reaction whatsoever; c) diphtheria anatoxine administered intravenously provoked mild congestion in the conjunctiva of the previously sensitized eyes; d) intravenous injection of antidiphtheria serum produced, 15 hours after administration, marked congestion of the conjunctiva of the sensitized eyes, lasting about 11 hours; the non-treated eye remained unchanged. From those treated with antidiphtheria serum, two animals received instillations of anatoxine and showed redness of the sensitized eye 15 and 24 hours later; the fellow eye remained normal. Intravenous injection of 2.5 cc. of anatoxine was followed by marked congestion of only the sensitized eye. (references) K. W. Ascher.

Nonnenmacher, H. **The influence of the weather on expulsive hemorrhages.** Klin. Monatsbl. f. Augenh. 123:45-52, 1953.

In the city hospital of Dresden three expulsive hemorrhages after cataract extractions occurred within 24 hours. This took place on the 27th and 28th of January, 1953. During that time the weather changed markedly and the temperature rose rapidly from −28° to +6° centigrade. It is quite likely that the sudden change of the weather precipitated the hemorrhages. (1 figure, 22 references)

Frederick C. Blodi.

3

VEGETATIVE PHYSIOLOGY, BIOCHEMISTRY, PHARMACOLOGY, TOXICOLOGY

Bishop, P. O., Jeremy, D., and Lance, J. W. **The optic nerve: properties of a**

central tract. J. Physiol. **121**:415-432, Aug. 28, 1953.

A technique of antidromic activation of the cat's optic nerve in situ was developed, using a modification of the Horsley-Clarke stereotaxic apparatus. All fibers in the cat's optic nerve are myelinated. The authors noted two groups of fibers, a group of smaller fibers which occupy all of the upper part of the tract and part of the lower, and a group of larger fibers found only in the lower part. They studied the axonal diameter and found it to range from 1 to 8.5 micra with occasional larger fibers. They also studied the peak conduction velocity of the crossed and uncrossed fibers and noted that the latter appeared faster, for some still unknown reason. They also studied the absolutely and relatively refractive periods, duration of supernormal period, amount and duration of negative after-potential, and the period of latent addition. They concluded that except for the duration of the supernormal period in the recovery of excitability following stimulation, all the properties of the optic nerve fibers studied are similar to those which are characteristic of the A fibers described in peripheral nerves by Grundfest and Gasser.

Harry Horwich.

Cascio, G., and Lodato, G. **Different absorption, in vitro, of nicotine by the retina and optic nerve.** Giorn. ital. oftal. **3**:253-259.

The effects of tobacco on the optic nerve and retina are extensively discussed, and various opinions quoted. In an attempt to determine the differential affinity for tobacco of both optic nerve and retina, the authors exposed these tissues to the same concentration of nicotine solution for the same period of time, and found that the optic nerve absorbs more nicotine than the retina. This leads the authors to the conclusion that in tobacco amblyopia, the primary lesion is in the optic nerve, and any changes found in the retina are secondary. V. Tabone.

Hassa, Abel. **Topical cortisone in eye disease.** Acta ophth. **30**:47-52, 1952.

The case histories of 20 patients with chronic ocular diseases treated with topical application of cortisone are analysed and reported in a statistical table. The author's experience confirms the general experience that cortisone is beneficial in inflammatory processes in the eye. He reports a rise in ocular tension following the instillation of cortisone in cases in which the tension was normal before cortisone therapy was instituted. The author has no explanation to offer for this phenomenon. He keeps his patient under cortisone therapy under daily control with the Schiøtz tonometer.

Ray K. Daily.

Kirber, H. P., and Kirber, M. W. **Transplantation of ocular tissues to the chorioallantoic membrane.** A. M. A. Arch. Ophth. **50**:2-15, July, 1953.

Tissue from human, guinea pig, rabbit and chicken eyes was kept alive when transplanted in chorioallantoic chick membranes. The tissues were inoculated with the viruses of vaccinia and herpes simplex. Characteristic inclusion bodies were formed within 24 to 72 hours. Such transplanted human tissues may prove a valuable means of studying viral diseases.

George S. Tyner.

Kornblueth, W., Yardeni-Yaron, E., and Wertheimer, E. **Glucose utilization of the retina.** A. M. A. Arch. Ophth. **50**:45-49, July, 1953.

Fresh rat retinas were placed in flasks containing various media with a known concentration of glucose. The tissues were incubated for two and one-half hours and the amount of remaining glucose was

determined. The glucose utilization of the retinas was thereby calculated. The influence of various organic and inorganic substances on glucose utilization was studied. Serum proved to be the best medium for glucose utilization of the retina. George S. Tyner.

Pannarale, M. R., and Leonardi, F. **Effect of isonicotinic acid hydracide on experimental ocular tuberculosis.** Boll. d'ocul. **32**:193-219, April, 1953.

Mycobacteria tuberculosis, human type, were inoculated into the anterior chamber of immunoallergic and of noninfected guinea pigs. Six animals received no therapy, six were given 10 mg./kg of INI for 30 days, six more were treated for 60 days, receiving the double dose during the second month of therapy; the injections were given in 1-percent aqueous solution and the animals were observed for more than three months. In immunoallergic animals the treatment was of preventive and of therapeutic value and effective even after discontinuation of administration. In nonimmunoallergic guinea pigs neither preventive nor curative results were registered. (10 photomicrographs, 7 graphs demonstrating the course of the infection, references) K. W. Ascher.

Salomaa, Salme. **Experiments with heparin.** Acta ophth. **30**:33-34, 1952.

Experimental investigations with topical applications of heparin to the eye show that a 30-percent solution causes a fall in intraocular pressure. The same solution injected subconjunctivally also causes slight miosis. Injected into the anterior chamber it produces a marked fall in the ocular tension which lasts 2 to 12 days. Ray K. Daily.

Salomaa, S., and Swanljung, H. **Clinical experience with ACTH and cortisone in eye diseases.** Acta ophth. **30**:53-58, 1952.

The results of 73 cases are tabulated. They confirm the results published in the literature. Ray K. Daily.

Sorsby, A., Ungar, J., and Crick, R. P. **Aureomycin, chloramphenicol, and terramycin in ophthalmology.** Brit. M. J. **2**: 301-304, Aug. 8, 1953.

The three drugs listed, when given systemically, do not penetrate readily into the eye. Chloramphenicol as a 15-percent suspension can be administered subconjunctivally and penetrates into the eyeball. Chloramphenicol as 1-percent ointment, or ½-percent drops, or by subconjunctival injections of a 15-percent solution gave gratifying results in 26 cases of epidemic keratoconjunctivitis. The author prefers chloramphenicol to aureomycin or terramycin. H. C. Weinberg.

4

PHYSIOLOGIC OPTICS, REFRACTION, COLOR VISION

Alpern, M., and Spencer, R. W. **Variation of critical flicker frequency in the nasal visual field.** A. M. A. Arch. Ophth. **50**:50-63, July, 1953.

There has been considerable confusion as to whether the critical flicker frequency for the center of the visual field is higher than, equal to, or lower than that for the peripheral field. The authors found that if variations in the size of the pupil were prevented from influencing the amount of retinal illumination the critical flicker frequency was successively less with increase in peripheral angle. With change in pupillary size the converse was true.

George S. Tyner.

Bahr, Gunnar. **Studies on the depth of focus of the eye.** Acta ophth. **30**:39-44, 1952.

The literature is reviewed and the experimental set-up of the investigation described. Because of technical difficulties

no conclusive data have emerged from the investigation. Ray K. Daily.

Berger, Curt. **The influence of stroke-width upon the legibility threshold of recognition of some narrow numerals of varying heights.** Acta ophth. **30**:409-419, 1952.

The influence on legibility of width and height of two numerals 0 and 5 with a constant stroke width was reported by Berger formerly. This report deals with an investigation on the influence of stroke-width of the same numerals, keeping the width constant at 1 mm. and varying the height from 2.5 mm. to 8.5 mm. The data show that increasing the retinal height had no signficant influence upon the recognition of the 0's. But increasing the retinal height of 5's increased the threshold of recognition significantly. Decrease in stroke-width from 0.25 to 0.1 mm. increased the legibility of 0's but did not affect the recognition of 5's. The theoretical significance of the data is discussed and the literature in this field reviewed. (5 tables) Ray K. Daily.

Cowan, A. **Monocular aphakia.** A. M. A. Arch. Ophth. **50**:16-18, July, 1953.

The visual axis is altered in aphakia so that it is necessary for the eye to diverge two degrees to obtain an image in the fovea. The retinal image in a corrected aphakic eye is thirty percent larger than in the normal eye. The removal of a senile cataract makes the eye many times more sensitive to violet and ultraviolet rays than before. An aphakic eye corrected for distance is blurred within four meters. Because of many factors brought about by removal of the lens, an aphakic eye cannot be made to function binocularly with a normal phakic eye. When single vision is obtained by correction of monocular aphakia, it is because of suppression.
George S. Tyner.

Epstein, L. Ivan. **The Luneburg distortion of Vieth-Mueller circles.** Am. J. Optometry **30**:463-466, Sept., 1953.

This is a discussion of the subjective horopter and the induced size effect of Ogle, suggesting experiments which could easily be performed.
Paul W. Miles.

Granstrom, K. O. **Eyes with normal visual acuity without glasses.** Acta ophth. **30**:107-114, 1952.

This is a report of the ocular examination of 1,254 applicants for the air force, aged between 18 and 25 years. The data relating to visual acuity, refraction, corneal astigmatism, heterophoria, stereoscopic vision, light sense, and incidental abnormalities are tabulated. (5 tables)
Ray K. Daily.

Hilding, A. C. **Studies of the otic labyrinth; on the deviation of the visual axes (as evidenced by the Maddox rod test) after the Bárány turning test.** Ann. Otol. Rhin. Laryng. **62**:462-469, June, 1953.

The author investigated the phenomenon of the deviation of the visual axes following vestibular stimulation by means of the Bárány turning test. The subjects were tested with a Maddox rod over one eye, use being made of a graduated tangent screen to evaluate the deviation. Measurements were made as a control before turning in the Bárány chair; immediately upon stopping the chair at the beginning of the period of post-rotational nystagmus and vertigo; at the end of that period; and in some cases, as a repeat test with the subject wearing small base-in prisms. In the control test, six showed convergence and three divergence. In the immediate postrotational period thirteen out of twenty-five showed a shift towards the convergence side; the remainder showed no change. At the end of that period the shift appeared to be toward divergence in fourteen out of twenty-four

cases, and no change in the remainder. The test with prisms before the eyes showed no significant changes. The author concludes that the deviation following the Bárány turning test as evidenced by the Maddox rod test does not appear to be a result of stimulation of the labyrinth, but rather a manifestation of the accommodation-convergence or fusion reflexes, or both. Harry Horwich.

Holm, Eiler. **Lental rainbow vision.** Acta ophth. **30**:35-37, 1952.

Holm describes the rainbow rings originating from the lens and calls attention to the fact that not all rainbow rings around a light are caused by glaucoma. He believes that they originate in the lens fibers. A method of demonstrating the rings is described. Ray K. Daily.

Kronning, Eric. **Recovery of vision after traumatic choroidal rupture through the macula.** Acta ophth. **30**:429-436, 1952.

This is a very interesting report of a case of contusion of the right eyeball in a five-year-old boy. The injury caused damage to the lens, a vitreous hemorrhage, and three large and one small choroidal ruptures. One large rupture passed right through the macula. About a month after the injury the visual acuity of the injured eye was 5/50, and the globe began to deviate outwards. Occlusion of the good eye led to recovery of visual acuity of the injured eye. A year after the injury vision in the injured eye was almost normal and binocular function was restored. Ray K. Daily.

Mattsson, R. **Transient myopia following the use of sulphonamides.** Acta ophth. **30**:385-396, 1952.

The literature on this phenomenon is reviewed, and two cases are reported. In both there was reduced accommodation, a shallow anterior chamber, and moderate mydriasis. In one of the cases there was marked chemosis of the conjunctiva. The author believes that the transient myopia is caused by an allergic edema of the ciliary body. Uneven distribution of this edema explains the apparently contradictory findings reported by various observers. Ray K. Daily.

Ogle, K. N. **Optics and visual physiology.** A. M. A. Arch. Ophth. **50**:81-108, July, 1953.

The pertinent literature is reviewed. George S. Tyner.

Ribas Bensusan, Jose. **The phases of sciascopy.** Arch. Soc. oftal. hispano-am. **13**:789-806, July, 1953.

The optics of sciascopy are analyzed. Ray K. Daily.

Siebeck, R. **Disturbance of space perception in anisocoria.** Klin. Monatsbl. f. Augenh. **123**:86-89, 1953.

Mydriasis or miosis in one eye only has the same effect as a gray filter in front of the smaller pupil. Patients with anisocoria will experience the Pulfrich phenomenon and may have difficulties in city traffic. (13 references) Frederick C. Blodi.

Ursin, K. V. **Experience with contact lenses made of plastic.** Acta ophth. **30**:11-18, 1952.

On the basis of answers to the questionnaire sent to eighty patients for whom contact lenses had been prescribed, Ursin concludes that the development of contact lenses is not yet satisfactory, that the fitting is time-consuming and troublesome, that many patients have difficulty in inserting their lenses, and that instruction in insertion should be given more attention. He found that patients who wear contact lenses for the correction of pathologic conditions such as keratoconus and irregular astigmatism, tolerate them better and persist in wearing them. Patients

wearing contact lenses for the correction of refractive errors reported more difficulties and less tolerance in their use. (4 tables) Ray K. Daily.

Walter, R. **The Moeller refractor for subjective refraction.** Klin. Monatsbl. f. Augenh. **123**:92-95, 1953.

This is a short addition to an article by Jensen (Klin. Monatsbl. f. Augenh. 121) who gave a detailed description of this modified Green refractor which is used with a projector chart. Only concave cylinders are used. This refractor enables speedier and more accurate refraction. (1 figure) Frederick C. Blodi.

Wieland, G. **Glare protection with polarizing filters.** Klin. Monatsbl. f. Augenh. **123**:89-92, 1953.

The author prefers polaroid glasses with the filter in the vertical direction as a protection against glare. (2 figures) Frederick C. Blodi.

5

DIAGNOSIS AND THERAPY

Beierwaltes, W. H. **X-ray treatment of malignant exophthalmos: a report on 28 patients.** J. Clin. Endocrin. **13**:1090-1100, Sept., 1953.

Thirteen patients out of 28 whose pituitary gland had been exposed to X rays showed a significant improvement in their exophthalmos. The average improvement amounted to almost 4 mm. and often was not apparent until seven months after the X-ray treatment. Along with this improvement the edema of the lid and of the conjunctiva decreased. Exophthalmos which has been present for more than two years did not respond satisfactorily to X-ray treatments. An area 8 × 7 cm. over each temporal area lateral to the pituitary was given a total of 1500 r per field. (5 figures) H. C. Weinberg.

Bellavia, M., and Pellegrino, F. **Retrobulbar injection of alcohol.** Giorn. ital. oftal. **3**:292-306, May-June, 1953.

The effect of retrobulbar injection of alcohol, notably on the reduction of ocular tension, on ocular pain and sensation, and on the optic nerve are extensively reviewed and discussed. Ten patients who were given 35 to 70 percent alcohol are described in detail. The results were satisfactory, the tension was reduced, and the inflammatory symptoms and photophobia were diminished or abolished. In some cases more than one injection was necessary to obtain the desired effect.

A few minor inconveniences due to this form of therapy are recorded, but it is stressed that no deterioration of the ocular condition ever followed this injection. V. Tabone.

Borioni, D. **Experiences with isonicotylhydrazide.** Boll. d'ocul. **32**:247-250, April, 1953.

Good results were obtained in severe cases of ocular tuberculosis, mainly in adult women. K. W. Ascher.

Caballero del Castillo, Daniel. **Our procedure for the diagnosis and localization of intraocular foreign bodies.** Arch. Soc. oftal. hispano-am. **13**:763-776, July, 1953.

As an identification guide for localization of intraocular foreign bodies, the author uses a ring with a stem, which is attached with sutures to the limbus. (13 figures) Ray K. Daily.

Holmberg, Ake. **The temperature of the eye during the application of hot packs, and after milk injections.** Acta ophth. **30**: 347-364, 1952.

This is an investigation on the effect of general hot packs and of milk injections on the temperature of the eye. The temperature was taken in the conjunctiva close to the limbus, with a copper constant thermo-element, as the patient lay in a hot pack, and after the milk injections. The tabulated data show that from

the standpoint of a rise of temperature the hot pack gives an inconstant and transitory effect. The rise of temperature is not significant and the maximum rise occurs immediately before the removal of the hot pack, and falls rapidly to its initial value. The temperature rose more in the eyes than in the mouth. Milk injections have a much greater although variable effect on the temperature of the eyes and mouth; the maximum temperature is reached between six and nine hours after the injection, somewhat earlier in the mouth than in the eye. (11 tables)

Ray K. Daily.

Michaels, David D. **The electrical responses of the eye.** Am. J. Optometry **30**: 451-462, Sept., 1953.

This is a review of the literature on the electroretinogram. It presents no new data. Paul W. Miles.

Pelkonen, Ulla. **On the administration of streptomycin and promizole in ophthalmic diseases.** Acta ophth. **30**:7, 1952.

Forty-two patients were given daily intramuscular injections of 1 gm. of streptomycin until a total dose of 30-45 gm. was administered. Sixty patients were given a similar dose of streptomycin and an oral dose of 6 gr. of promizole internally. The results in the two groups were alike. In iritis, iridocyclitis and uveitis streptomycin shortens the duration of the disease, but does not fully cure the process itself, and relapses occur and are severe. No significant effect was seen in cases of sclerokeratitis, and no effect whatsoever in cases of retinal periphlebitis.

Ray K. Daily.

Regard, E. **Pressure scissors for placental implants.** Ann. d'ocul. **186**:221-24, March, 1953.

The author has enlarged the ordinary DeWecker scissors making the blades heavier with a sharp edge externally. This modification is suggested to facilitate dissection of the subconjunctiva and section of dense tissues such as the sclera. A smaller incision is also frequently possible, reducing the number of sutures necessary for closure. Chas. A. Bahn.

Riise, Per. **The international standardization of visual field charts.** Acta ophth. **30**:105, 1952.

Riise urges the adoption of international visual field charts. Ray K. Daily.

Schillinger, R. J. **An aid in ocular anesthesia.** A. M. A. Arch. Ophth. **50**:78, July, 1953.

To prevent discomfort, a mixture of three drops of 5-percent cocaine solution and one drop of 1:1,000 adrenalin is instilled topically before placement of a superior rectus suture. The mixture is placed high in the superior conjunctival cul de sac with a syringe and long lacrimal canula. George S. Tyner.

Swan, K. C. **A dehydrating jelly to clear corneal bedewing.** A. M. A. Arch. Ophth. **50**:75-77, July, 1953.

A stable jelly for office use in dehydrating corneas may be prepared by adding 2 grams of sodium carboxymethylcellulose to 100 cc. of U.S.P. glycerine. The preparation is administered in a standard ophthalmic ointment tube. Topical anesthesia should be instilled before its use and the jelly confined to the cornea.

George S. Tyner.

Thiry, S., and Lavergne, G. **Angiography of the orbit.** Acta ophth. **30**:379-383, 1952.

The authors describe the technique of angiography, and the appearance of the orbit with the vessels injected with radioopaque material. It is shown that this method of examination reveals the position and the caliber of many orbital arteries, and visualizes the changes of the veins in arterio-venous aneurysms. While this diagnostic procedure is still in

its inception, it appears to offer aid in the diagnosis and localization of orbital tumors. (12 figures) Ray K. Daily.

Vannas, M., Sallinen, I., and Sysi, R. **X-ray treatment of tuberculous diseases of the eye.** Acta ophth. **30**:5-6, 1952.

Of 133 cases treated, 116 responded to this form of therapy, the best results being obtained in tuberculous sclerokeratitis, iridocyclitis, and retinochoroiditis. The treatment consists of four treatments of 50 to 100 R, at intervals of 3 to 4 days. The filter was 3 mm. of aluminum and the distance 40 cm. A course of treatment was repeated after four weeks.

Ray K. Daily.

Vannas, Mauno. **Needle forceps fixation of the eye.** Acta ophth. **30**:123, 1952.

The instrument described consists of two surgical needles, attached with screws to a forceps; the needles are changeable and their position adjustable.

Ray K. Daily.

6

OCULAR MOTILITY

Adler, F. S. **Pathologic physiology of strabismus.** A. M. A. Arch. Ophth. **50**:19-29, July, 1953.

The author presents experimental evidence that divergence from the convergent position is associated with active contraction of the external rectus muscles. The controversy over whether a divergence mechanism exists or whether divergence is simply the result of relaxation of the convergence mechanism is thereby settled—a divergence mechanism exists. A brief, but lucid explanation of the neuromuscular mechanisms which control eye movements is presented. Certain conclusions regarding the pathologic physiology in strabismus follows the description of the normal physiology.

George S. Tyner.

Halbron, P. **The examination of ocular motility with complimentary colors.** Ann. d'ocul. **186**:202-216, March, 1953.

Ocular motility tests based on complimentary colors are considered superior because of greater accuracy and practicality. The primary purpose of this contribution is to promote a better understanding and wider use by French ophthalmologists of the well-known Lancaster test and Hess chart in combination, which the author prefers. After a description of the necessary equipment, the author presents illustrative cases with diagrams of the phorias, tropias and types of prolapses.

Chas. A. Bahn.

Leydhecker, W. **Divergence paralysis.** Klin. Monatsbl. f. Augenh. **123**:83-86, 1953.

A typical case in a 63-year-old man who suffered a contusion of the brain after a severe fall is presented. Immediately afterwards diplopia was noticed. The images had the same distance in all directions of gaze and the diplopia disappeared on convergence. A traumatic lesion of the divergence center is assumed. (39 references) Frederick C. Blodi.

Pisano, E. **Anterior internuclear paralysis of luetic origin.** Boll. d'ocul. **32**:242-246, April, 1953.

A twenty-five-year-old woman had a mild optic neuritis in her left eye in June, 1948, which soon healed. Three years later she complained of diplopia which proved to be due to anterior internuclear paralysis. Complete recovery was achieved by bismuth and penicillin administration, with recovery of the previously typical spinal findings as well as the ocular lesion. (references) K. W. Ascher.

Wheeler, M. C. **Strabismus.** A. M. A. Arch. Ophth. **50**:109-122, July, 1953.

The pertinent literature is reviewed.

George S. Tyner.

7
CONJUNCTIVA, CORNEA, SCLERA

Barraquer, Cerrero Tomas. **Total conjunctival flap for infected ulcers of the cornea.** Arch. Soc. oftal. hispano-am. 13:682-683, June, 1953.

The literature is reviewed and a case is reported. A patient in a poor state of nutrition had a corneal ulcer which resisted intensive treatment with antibiotics. The eye seemed to be lost. After cauterizing the ulcer, the cornea was covered with a complete conjunctival flap. Four days later, when the sutures were removed, the cornea was found transparent and the conjunctiva adherent to the base of the ulcer. The conjunctival pedicle was subsequently separated. This was followed by atrophy of the conjunctival vessels, leaving the entire corneal surface transparent. (1 figure) Ray K. Daily.

Broendstrup, Paul. **Infantile cystinosis.** Acta ophth. 30:365-377, 1952.

Two cases of this rare metabolic disease are reported. It is characterized by arrested growth, anorexia, polyuria, polydipsia, and rickets. The occurrence and finding of crystals in the cornea and conjunctiva makes the otherwise difficult diagnosis easy. The diagnosis of cases with suspected cystinosis may be established within a few minutes by the opthalmologist if he finds the tiny light reflecting granules over the corneal parenchyma, which give the cornea a dusty impression. The crystals are also found in the conjunctiva which like the cornea appears to tolerate their presence without reactive phenomena. (4 photomicrographs)
Ray K. Daily.

Busacca, A. **Pathology and therapy of trachoma.** Rev. Intern. du Trachome 30: 209-253, 1953.

This comprehensive study is illustrated with 46 fine illustrations. Infiltration and edema induce both fibroblastic proliferation, as seen in papillary hypertrophy, and the destruction of the adenoid structures with replacement by cicatricial connective tissue. The scar-like stripes seen clinically represent condensations of the cicatricial layer. The advent of the cicatricial stage is accompanied by progressive shrinking and disappearance of the hypertrophied follicles. The trachomatous process begins in the conjunctiva, but spreads in some cases to the pretarsal tissue, tarsus, orbicularis and smooth muscle. The contracted tarsus exerts pressure on the anterior segment of the bulb impeding corneal nutrition, thus causing the dystrophic type of pannus. The crimpling of the tarsus and the accompanying cicatricial retraction of the conjunctiva determines entropion. James E. Lebensohn.

Cassady, J. V., and Culbertson, C. S. **Cat-scratch disease and Parinaud's oculoglandular syndrome.** A. M. A. Arch. Ophth. 50:68-74, July, 1953.

Four cases of Parinaud's oculoglandular syndrome are reported. In each case the patient showed a positive skin test to the antigen of cat-scratch disease.
George S. Tyner.

Church, R. E., and Sneddon, I. B. **Ocular pemphigus.** Brit. J. Derm. 65:235-245, July-Aug., 1953.

Of the seven cases reported six were in women with an average age of 67 years. Lesions appeared in the conjunctiva, mouth, nose, and genitals. Mucosal lesions and conjunctivitis may precede pemphigus vulgaris and pemphigus vegetans by many months. The differential diagnosis between mucosal pemphigus and the other pemphigus eruptions may be confirmed by demonstrating histologically that the intact bulla of mucosal pemphigus is subepidermal. Evidence from these cases and the literature is presented to uphold the concept that ocular

pemphigus is a disease entity separate from all the other members of the pemphigus group and that it never ends by transition to pemphigus vulgaris. (5 figures) H. C. Weinberg.

Gonzalez Vanrell, Francisco. **Conjunctival autoplasty for pterygium surgery.** Arch. Soc. oftal. hispano-am. 13:631-642, June, 1953.

In ten cases of pterygium, the author used autotransplants of conjunctiva with satisfactory results. The technique consists in the complete excision of the pterygium, and covering of the raw surface by a quadrangular flap of conjunctiva excised from the supero-external portion of the globe. The graft is anchored to the sclera with four silk sutures, and the operation terminated by two conjunctival sutures which unite the margins of the conjunctival wound in the upper portion of the eyeball. (12 figures)
Ray K. Daily.

Gronvall, Herman. **The report of a case of vaccinial disciform keratitis treated with cortisone.** Acta ophth. 30:59-61, 1952.

A 15-year-old boy developed a vaccinial keratitis of the right eye 12 days after being vaccinated for small-pox. The eye improved rapidly upon the institution of topical applications of cortisone. (2 figures) Ray K. Daily.

Junceda Avello, J. **A technique for the insertion of preplaced sutures in penetrating keratoplasty.** Arch. Soc. ophtal. hispano-am. 13:659-664, June, 1953.

The technique described consists in inserting four sutures into the graft while it is still in place, trephining the host cornea to half of its depth, and staining the groove with fluorescein, and passing the ends of the four sutures in the graft through the margins of the wound in the host cornea which at this point is not completely perforated. When the four sutures are inserted, trephination is completed, and the sutures tied. This procedure adds safety to the operation, by doing away with the requirements for perfection in the sharpness of the instruments, but eliminating trauma to the margins of the host's cornea which need not be touched with forceps, and by eliminating pressure incident to the insertion of sutures in an open eyeball. (4 figures)
Ray K. Daily.

Kohn-Kony, M., and Raphael, E. R. **On trachoma treated solely by antibiotics.** Rev. Intern. du Trachome 30:280-291, 1953.

Trachomatous children treated only with antibiotic ointments applied locally showed healing in 12 to 18 months. Auxiliary use of scraping or the copper sulphate stick irritated and aggravated the condition. James E. Lebensohn.

Lorente, J. **Changes in the corneal nerve fibers in a case of Groenow's dystrophy of the macular type.** Arch. Soc. oftal. hispano-am. 13:746-762, July, 1953.

This is a detailed report of the anatomic-pathologic examination of the corneal section obtained in the course of a keratoplasty in a case of Groenow's hereditary dystrophy. The microscopic sections show a conservation of the epithelium and subepithelial plexus, and profound degenerative changes in the nerve fibers of the corneal parenchyma; these findings support the neurogenic theory of the etiology of this disease. The author reviews the literature on the anatomy and physiology of the corneal innervation, on the anatomic studies of corneal dystrophies, and on the theories relative to the etiology of this disease. (10 photomicrographs) Ray K. Daily.

Oksala, Arro. **Interstitial keratitis and chorioretinitis.** Acta ophth. **30**:437-441,

The use of cortisone in a case of interstitial keratitis cleared the cornea sufficiently to permit a study of the fundus. At the onset of the disease white perivascular streaks following the medium-sized veins were found in the right fundus. About four months after the onset a black chorioretinal patch appeared in the same eye. The author holds that interstitial keratitis is actually a kind of panophthalmitis, and that formerly the deeper lesion escaped discovery because of opacity of the cornea. It is possible that the uvea may be the primary seat of the disease. Cortisone therapy in this case had a rapid but transitory effect, and the symptoms became greatly aggravated each time cortisone was discontinued.

Ray K. Daily.

Orzalesi, F. **First trials of lysozyme in herpetic keratitis.** Giorn. ital. oftal. **3**:210-216, May-June, 1953.

Previous experiments by the author had shown that lysozyme was capable of inactivating in vitro the virus of herpes and that there was a clear relationship between the lysozyme content of the blood, tears and cornea and immunity to the herpetic infection. Orzalesi had also shown that substances like cortisone, which had an adverse effect on herpetic infection, actually reduced lysozyme activity in the blood and secretions. With these facts in mind he now treated fifteen human cases of superficial herpetic keratitis with this ferment. Drops of 2-percent lysozyme (Armour) were instilled every 4 to 6 hours, in addition to the usual mydriatics. In some cases light scraping of the base and margins of the ulcers was also done, followed (once only) by the instillation of penicillin drops. Lysozyme was then continued till healing occurred. In 14 cases the eyes recovered in 5 to 8 days, while one other case appeared to get worse, and treatment was suspended after only three days. Lysozyme produced no unpleasant symptoms, and no irritation during the whole period of treatment, which never exceeded eight days. In one case only after the eighth day there appeared an allergic conjunctivitis, which disappeared quickly on cessation of treatment. V. Tabone.

Salvi, G. L. **Unusually located bilateral primary band-shaped keratosis.** Boll. d'ocul. **32**:225-232, April, 1953.

A 74-year-old man, otherwise in good health, complained of poor vision in both eyes for some years previous to admission. In the center of both corneas a superficial, grayish brown, irregularly shaped avascular 5 by 3 mm. large opacity was found. The epithelium and Bowman's membrane were involved while the stroma, Descemet's membrane and endothelium were clear. A reduction of corneal sensitivity was restricted to the lesion. The etiologic factors and localisation are discussed. (drawing of cornea, photomicrograph, references) K. W. Ascher.

Sjøgren, H., and Eriksen, A. **ACTH and keratoconjunctivitis sicca.** Acta ophth. **30**:463-465, 1952.

ACTH was tried in four cases of keratoconjunctivitis sicca, without any appreciable change in the clinical picture.

Ray K. Daily.

Tsutsui, J. **Isolated corpuscles of trachoma.** Rev. Intern. du Trachome. **30**:307, 1953.

The corpuscles that apparently represent the virus of trachoma were purified by differential centrifugation and photographed with the electron microscope (× 20,000) as illustrated. Inoculation studies

with this "pure virus" have yet to be done.

James E. Lebensohn.

Wegener-Thompson, S. **Deliberations about the possibility of a corneal prosthesis of plastic.** Acta ophth. 30:9-10, 1952.

This is a preliminary report on the possibility of using plastic to replace a portion of an opaque cornea. The author describes the plastic lens which he designed, but has not as yet used.

Ray K. Daily.

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

Latte, B. **A case of recurrent hypopyon iritis.** Giorn. ital. oftal. 3:283-291, May-June, 1953.

The author describes in detail a patient who had bilateral iridocyclitis and conjunctivitis, and responded to aureomycin therapy. Though there was no real hypopyon, the aqueous was very turbid; the patient also had fever, had articular pains, and showed extensive aphthous stomatitis. In an attempt to establish the viral origin of the affection, material from aphthous ulcers and from the aqueous were inoculated into the chick embryo, with completely negative results. The hemoagglutination and complement deviation tests on the patient's serum during an acute stage of the disease, using amniotic fluid and chorio-allantoic membrane as antigen, were negative, as also was the virus interference test.

V. Tabone.

Murray, R. G. **Studies of the aqueous exudate in uveitis.** Canad. M.A.J. 68:124-126, Feb., 1953.

Twenty-two cases of non-granulomatous uveitis were investigated to see if the aqueous contained bacteria or other organisms, to count and classify the cells found in the aqueous, and to correlate, if possible, these findings with the clinical picture. No organisms were found on smear, and only one culture was positive, probably as a result of contamination. Electron microscope studies also were negative. Cytologic examination showed lymphocytes and neutrophilic polymorphonuclear cells from the blood stream, and chromatophores and macrophages from the tissues. There was also debris, many ghost cells and a few corneal endothelial cells. The tissue cell response varied inversely with the total cell count. In one case associated with a severe rheumatoid arthritis there was a high relative eosinophile count in the aqueous (10 percent). This patient was resistant to all therapy including the use of cortisone and ACTH. In 17 out of 20 cases of endogenous uveitis the response was predominantly lymphocytic; in the two of exogenous inflammation it was polymorphonuclear. The cell count and cell types were not related to the duration of the illness, and varied widely, apparently being proportionate only to the density of the aqueous observed biomicroscopically.

Harry Horwich.

9

GLAUCOMA AND OCULAR TENSION

Alajmo B., and Simonelli M. **Perforating cyclodiathermy for glaucoma, especially infantile glaucoma.** Giorn. ital. oftal. 3:205-209, May-June, 1953.

After a brief review of the way cyclodiathermy acts in glaucoma, the authors submit results of this method of treatment in 42 cases. These included infantile and juvenile types, secondary glaucoma from various causes, glaucoma after iridocyclitis, hemorrhagic and absolute glaucomas, and cases of persistently raised intraocular pressure after fistulizing operations. The authors used a 2-mm. needle, and made the perforations through the conjunctiva, at a distance of 3 mm. from the limbus. When only the superior

quadrant was operated on, the number of perforations varied from 8 to 10, while 18 to 20 perforations were carried out, if it was desired to cover the whole circumference of the ciliary body. Cases were followed from three months to two years, and results were classified as "good" when the tension was brought down to normal, and remained in this condition during the whole period of observation, "good but transitory," when the initial good results were not maintained, and "negative" when no response was shown. The results obtained were the following: 45 percent good, 40 percent good but transitory, and 15 percent negative. The authors believe that the best results of perforating cyclodiathermy are to be expected in cases of infantile glaucoma and of iridocyclitis with increased intraocular pressure. The authors have also found the procedure useful in those forms of secondary glaucoma, such as hemorrhagic and absolute, which do not respond well to other forms of treatment. V. Tabone.

D'Ermo, F. **Chronic glaucoma and keratoconus.** Boll. d'ocul. **32**:220-224, April, 1953.

Two patients with these diseases, aged 64 and 70 years, were observed in the Eye Clinic of Rome. The condition was bilateral in both and complicated by nuclear cataract in one of them. Keratoiridectomy in one, and trepanation in the other patient gave good results. The coincidence of keratoconus and glaucoma is rare and the previous publications and possible relationship are discussed. (references) K. W. Ascher.

Funder, W. **The visual field in partial glaucomatous excavation.** Klin. Monatsbl. f. Augenh. **123**:16-19, 1953.

The influence on the visual field of a cup which reaches the margin in one area only has been interpreted in various ways. In 23 eyes the excavation was temporal and up, in 25 temporal and down. The field defects were correspondingly in the nasal lower and in the nasal upper quadrant. In four eyes the glaucomatous excavation reached the margin of the disc only at the nasal side. There was a concentric constriction in the temporal field in all these patients and in two of them there was an additional scotoma from the blind spot to the temporal side. (3 figures, 9 references) Frederick C. Blodi.

Gonzales-Pola, Angel Moreu. **Surgery of glaucoma.** Arch. Soc. oftal. hispano-am. **13**:707-730, July, 1953.

The author warns against confounding glaucoma with ocular hypertension which is the factor combatted by surgery. He divides glaucoma procedures into fistulating and nonfistulating. Of the fistulating types he prefers the Elliott trepanation, considering that iridencleisis has no advantages. He considers fistulating operations indicated in the presence of inadequate drainage, evidenced by a poor state of the uvea, poor pupillary reactions, persistent and high tension, a partial or completely blocked angle of the anterior chamber, poor elimination of fluorescein, and poor response to nicotinic acid. The nonfistulating operations are indicated when the phenomena of hypersecretion dominate the clinical picture; these are a good state of the uvea, good pupillary reactions, episodes of moderate hypertension, a free angle of the anterior chamber, good elimination of fluorescein, and a good response to nicotinic acid. The author performs cyclodiathermy through the anterior chamber under the control of a gonioscopic lens, a method which he described in 1944. Ray K. Daily.

Hofmann, H., and Holzer, H. **The effect of muscle-relaxing drugs on the intraocular pressure.** Klin. Monatsbl. f. Augenh. **123**:1-16, 1953.

This is a comparison of the effect of

curare and two synthetic muscle relaxants on the eye. The relaxing effect is stronger and longer-lasting with curare. The first symptom is crossed diplopia and then ptosis. Under curare the intraocular pressure is lower than before. The intraocular pressure changes little with Decamethonium. After the bis-cholic ester of succinylic acid the intraocular pressure rises because of contracture of the external eye muscles. (13 figures, 75 references) Frederick C. Blodi.

Marin-Amat, M. **The mode of action and value of iridencleisis.** Arch. Soc. oftal. hispano-am. **13**:610-630, June, 1953.

The author traces the history of glaucoma surgery to the present status of the various antiglaucomatous operations in the various countries. This is followed by a review of the literature on the modern conception of the etiology and surgical therapy of glaucoma. The article is concluded by a brief review of the author's four publications which outline his conception of the pathogenesis and the mechanism of action of antiglaucomatous operations. He maintains that the intraocular circulation is regulated by the neurovegetative system, the principal center of which is autonomous and is located in the ocular wall; it is formed by the small nerve ganglia of Krause and Müller, situated in the ciliary body and choroid, respectively. The primary process in glaucoma is an increase in blood volume and increased production of aqueous; as a consequence a greater or lesser obstruction to the drainage of aqueous develops. Surgical procedures in glaucoma should aim primarily to modify the production of intraocular fluids. The inclusion of the iris is the principal factor in the mechanism of reduction of the ocular tension; it acts on the circulus iridis major of the iris and the sympathetic plexus which surrounds it; it acts by a sedative effect on the neurovegetative excitability, and thereby restricts the blood supply to the ciliary processes; the diminished excretion which follows accounts for the fall in intraocular pressure. The iris inclusion thus acts as a true sympathectomy and arteriectomy. The effect of fistulization is secondary, augmenting the primary effect, in case the reduction in the production of aqueous is inadequate.

Ray K. Daily.

Posner, A., and Schlossman, A. **Further observations on the syndrome of glaucomatocyclitic crises.** Tr. Am. Acad. Ophth. **57**:531-535, July-Aug., 1953.

The syndrome of glaucomatocyclitic crises is presented as a clinical entity which should be differentiated from other types of glaucoma. It consists of recurrent attacks of acute glaucoma associated with signs of mild cyclitis. During an attack, the eye is white and pain is minimal despite marked elevation of tension. A few keratic precipitates are present and the pupil of the affected eye is larger than the other. The attack is self-limited. Visual acuity and field remain unimpaired. The angle of the anterior chamber is open, even at the height of the attack. This differentiates it from narrow angle glaucoma. Between attacks, all provocative tests give normal responses and facility of outflow is normal. Treatment should be limited to mild miotics and topical cortisone. No treatment is necessary in the interval between attacks. Surgical intervention does not prevent recurrences. Theodore M. Shapira.

Sokolic, P. **Nonperforating cyclodiathermy combined with cyclodialysis.** Klin. Monatsbl. f. Augenh. **123**:30-34, 1953.

A nonperforating diathermy is done in the upper segment after the conjunctiva has been dissected. A ball electrode of 1½

mm. is used for one second. A cyclodialysis is done with an incision 2 mm. away from the limbus at 12 o'clock. This operation was performed on 54 eyes of 40 patients with various forms of glaucoma. The tension could be normalized in all of them. (2 figures) Frederick C. Blodi.

Vannas, M., and Bjorkenheim, B. **On hypotony following cyclodialysis, and its treatment.** Acta ophth. **30**:63-64, 1952.

Three cases of postoperative hypotension are reported. Gonioscopy demonstrated large clefts between the sclera and ciliary body. Attempts to close the clefts and normalize tension were made in all three cases. In two the ciliary body and sclera were electrocoagulated from the inner side, after making an incision into the anterior chamber. One patient developed an acute glaucoma ten days after the operation. The second patient died of cardiac failure and the histologic examination of the eye revealed a large space between the sclera and the ciliary body. In the third eye, which was aphacic, an unsuccessful attempt to close the cleft was made by electrocoagulation applied externally; the detached part of the ciliary body was then sutured to the sclera with three silk sutures. A scleral staphyloma, developed at the site of the sutures, was excised a half a year later. Tension was normalized, and vision improved from counting fingers at one meter to counting fingers at three meters.

Ray K. Daily.

10

CRYSTALLINE LENS

Arruga, H., and Arruga, A. **Results of cataract extraction, with the inclusion of Ridley's lens.** Arch. Soc. oftal. hispano-am. **13**:731-745, July, 1953.

The authors describe two forceps which they devised; one for grasping the Ridley lens, and the other for maneuvering the iris in front of the lens. The results of 23 operations are tabulated in detail. They conclude that the chief indication for this operation is a mature unilateral cataract. The technique is delicate and the extracapsular extraction is preferable. The operation involves a proliferation of iris pigment, which retards visual recovery. Tolerance of the lens is perfect. The results on the whole are favorable. (1 table, 3 figures) Ray K. Daily.

Cogan, D. G., and Dreisler, K. K. **Minimal amount of X-ray exposure causing lens opacities in the human eye.** A.M.A. Arch. Ophth. **50**:30-34, July, 1953.

Human subjects who have been exposed to a know amount of radiation were examined in an effort to determine the least amount of radiation which can produce opacification of the lens. All patients who received 800r (200 K.V.) or more through the eye showed opacifications of the lens. One of three patients who received 800r (200 K.V.) showed posterior subcapsular opacities. This total amount of radiation was received in three successive days as treatment for a conjunctival lymphoma. George S. Tyner.

Dellaporth, A. **Wound healing after cataract extraction.** Klin. Monatsbl. f. Augenh. **123**:65-74, 1953.

This is a comparison of the disadvantages and advantages of a few types of sutures. Conjunctival sutures are regarded as inadequate. Two types of corneo-scleral sutures are discussed, the McLean (Mendoza) and the Stallard (Leigard) type. The author prefers the latter because of its simplicity. (2 figures, 4 references) Frederick C. Blodi.

Dolcet, Louis. **A report of experience with Ridley's lens.** Arch. Soc. oftal. hispano-am. **13**:665-571, June, 1953.

Seven cases are reported. To facilitate visibility of transparent lens masses the author uses an ultraviolet filter on a head lamp. In one case the Ridley lens was dislocated, but well tolerated. In two cases it was removed, in one immediately after its insertion because there appeared to be a rise in ocular tension, and in the other case postoperatively because of a postoperative uveitis. In one case the result was not determined because the patient died. In two there was seclusion of the pupil with vision of 0.1 In one the result was satisfactory with vision 1/6. The poor results are attributed by the author to the poor condition of the eyes, and not to the surgical technique. Ray K. Daily.

Friede, R. **Cataract extraction by suction.** Klin. Monatsbl. f. Augenh. **123**:74-83, 1953.

The author has designed a cannula with which soft lens material can be sucked out of the eye. The suction is regulated by a rubber tube which the operator holds in his mouth. This method proved to be of some advantage in cataracts of young patients or after extracapsular extraction. (3 figures) Frederick C. Blodi.

Fry, W. E. **Pathologic findings in an eye with a nonhealing cataract incision.** Tr. Am. Acad. Ophth. **57**:537-538, July-Aug., 1953.

The history and important pathologic findings of a case in which a cataract incision failed to heal are recorded. The patient showed no unusual constitutional changes and there were no complications due to technique at the time of operation or subsequently at the time of repair. The patient was cooperative and there was no history of trauma to explain the nonhealing. The only pathologic finding of importance was the presence of hemorrhage in the subchoroidal space which was not massive. The apparent cause for this nonhealing was a subacute subchoroidal hemorrhage.

Theodore M. Shapira.

Giardini, A., and Nizetic, B. **Clinical and statistical study of postoperative hemorrhages after cataract extraction, in relation to capillary fragility.** Giorn. ital. oftal. 3:260-263, May-June, 1953.

The varieties and types of hemorrhage encountered after cataract surgery are reviewed, as well as the possible etiological factors. The capillary fragility was tested in 142 patients with senile cataract by the Gothlin method, but no significant relation between capillary fragility and postoperative bleeding was found. The view is expressed that the most likely cause for such hemorrhage is a mechanical one.

V. Tabone.

Hartmann, E., and Lombard, G. **Preoperative mydriasis and postoperative myosis.** Ann. d'ocul. **186**:193-201, March, 1953.

The primary purpose of preoperative mydriatics, especially in cataract extraction, is to facilitate the mechanical removal of the lens through a widely dilated pupil. As mydriatics of the atropine, homatropine and scopolamine group become inactive when the aqueous escapes, they are of little practical value because the pupil is not dilated when the lens is subsequently extracted. Mydriatics of the adrenalin, neosynephrine type, on the contrary, do not lose their effectiveness when the aqueous escapes and, therefore, are preferable. The most desirable preoperative mydriatic is a 5-percent neosynephrine solution instilled 20 minutes before operation and also immediately before operation, if necessary. The subconjunctival injection of 1 to 1,000 adrenalin accomplishes practically the same results.

Postoperative myotics are employed primarily to prevent peripheral synechiae

and iris prolapse. If retrobulbar anesthesia or cocaine has been used, a pilocarpine solution injected into the anterior chamber is preferable because eserine instillation does not begin to act until the anesthesia has ceased. Chas. A. Bahn.

Llorca, Perez. **The advantages and disadvantages of a corneal section with keratome and scissors in cataract extraction.** Arch. Soc. oftal. hispano-am. 13:603-609, June, 1953.

Llorca advocates instruction in the technique of a keratotomy with keratome and scissors, maintaining that a keratotomy made in this fashion is more accurate in position, freer of complications, and less exacting of surgical skill.

Ray K. Daily.

Poyales, A. **Elimination of the crystalline lens for the correction of high myopia.** Arch. Soc. oftal. hispano.-am. 13:777-787, July, 1953.

Poyales reviews the literature on Fukala's operation, and reports in tabulated form the data relating to preoperative and postoperative refraction, preoperative and postoperative visual acuity, technique of the operation and complications of 103 operations. Forty-six of the operations were extractions and fifty-seven discisions. He advocates a revival of this procedure, which has been largely abandoned.

Ray K. Daily.

Rizzini V. **A contribution to the study of "Ringstarlinse" of Von Szily (umbilicated cataract).** Giorn. ital. oftal. 3:217-229, May-June, 1953.

An extensive review of the published cases, and of the opinions expressed on congenital annular or umbilicated cataract, is given. Various opinions on the etiology and mode of transmission are quoted, and the author believes that aplasia or hypoplasia of the lenticular nucleus is at the basis of this type of cataract. He then describes two cases seen by him at the University Eye Clinic at Geneva, in one of which changes occurred in both eyes. He advises surgical treatment, and expresses the opinion that discission followed by removal of lens matter is to be preferred to total lens extraction. V. Tabone.

Silvan Lopez, F. **Suction cup for cataract extraction and insertion of Ridley's lens.** Arch. Soc. oftal. hispano-am. 13:672-681, June, 1953.

This a preliminary report on the construction of a suction cup made entirely of rubber. Silvan designed it to increase its suction power. A cup made entirely of rubber exerts a suction provided by the vacuum and by capillarity. He describes the various models he designed in search for a perfect model. The result is a cup which is applicable to cataract extraction, and which facilitates the introduction of the Ridley lens. (4 figures)

Ray K. Daily.

11

RETINA AND VITREOUS

Appelmans, M., and Lebas, P. **Angioid streaks and associated lesions.** Ann. d'ocul. 186:225-246, March, 1953.

Angioid streaks are primarily a degeneration of the outer or inner layer of Bruch's membrane or both. This membrane, like others which often separate basically different tissues throughout the body, is the secretion of both adjoining tissues and is subject to degenerations involving the elastic membranes throughout the body. After a lineal weakening of Bruch's membrane which usually radiates from the disc, hematinic pigment is deposited from the pigment epithelium or the choroidal pigment. Not infrequently there is an associated vascular degenera-

tion with resulting hemorrhages, especially of the small vessels which form the circle of Zinn. Later, cholesterin deposition and other degenerative changes occur which tend to make the lesions progressive. The same condition in the skin is termed "pseudo-xanthoma-elastica," and in the layer of Bowman the process is termed the syndrome of Groenblad and Strandberg. Chas. A. Bahn.

Bottoni, A., and Dugnani, E. **Small venous dilatations in diabetic retinopathy.** Giorn. ital. oftal. **3**:230-252. May-June, 1953.

The authors believe that the small, discrete, roundish punctiform red spots, seen preferably around the disc and macula, are the basis of all the other retinal manifestations in diabetes. The various etiologic theories are discussed, and the varieties of these small venous aneurysms are shown by means of several beautiful pictures. It would appear that they represent the effect of a toxic substance circulating in the blood of diabetics, acting on that part of the vessel wall exposed to the greater friction (bifurcation of capillaries, or sharp turning of same). The three possible methods of formation of these vascular alterations are, 1. Rupture of vessel wall, and encapsulation of the small resulting hemorrhages, 2. giving way of the altered wall of the vessel and the formation of a real aneurysm, and 3. preexistent, or newly formed loop of a capillary, with absorption of the two adjacent walls of the loop, thus formation of a vascular sac. On the basis of their own research, the authors favor the second theory. Commenting on the finding by Ashton and others, of hyaline deposits in the capillary walls of diabetics, and in the intercellular space of the islands of Langerhans, the view is advanced, that deposition of this hyaline substance is the cause of the symptoms of diabetes.

V. Tabone.

Lerner, H. A. **Surgical management of retinal detachment with retinal tears in retrolental fibroplasia.** A.M.A. Arch. Ophth. **50**:64-67, July, 1953.

Retinal tears were found in two premature infants with retrolental fibroplasia. The tears were successfully sealed surgically. Aside from the retinal tears the infants presented the usual clinical picture of retrolental fibroplasia.

George S. Tyner.

Lijó Pavía, J., and Lachman, R. **Macula. Crystalline tapeto-retinal degeneration observed over 11 years.** Rev. oto-neuro-oftal. Sud-am. **28**:59-67. May-June, 1953.

This is a third in a series of papers published by Lijó Pavía about crystalline luminescent deposits situated mostly in the macular area and which he interpreted as cholesterol crystals. At the time of publication of the two previous papers it was thought that they were reversible lesions and they were classified as acquired tapeto-retinal degenerations. The authors now describe in detail the ophthalmoscopic findings in another subject who had the same type of crystalline-luminescent deposits in the macular area of both eyes without any change for seven years. When the patient was seen again four years later, the luminescent deposits had disappeared and there was an area of retinal and choroidal atrophy, much more extensive than the original lesion, surrounded by white and yellowish spots. The lesion must be differentiated from disciform macular degenerations, "honeycomb" choroiditis, central pigmentary degeneration, luetic retinochoroiditis and Stargard's disease. The authors believe that this is a tapetoretinal degeneration but not a congenital one. Because there is no indication of a familial character, they assume that the lesion is probably the result of an acquired degenerative process. (4 figures, 8 references)

Walter Mayer.

Rosengren, B. **300 cases operated for retinal detachment. Method and results.** Acta ophth. **30**:117-122, 1952.

This is an analysis of the histories of 300 patients with retinal detachment, operated upon between 1936 and 1950. The data relating to the number and position of the ruptures, the age of the detachment, the method of operation, the postoperative visual acuity, and the complications are tabulated. Rosengren is a strong advocate of air injections into the vitreous in eyes with high retinal elevation. The table on visual results shows that good results may be obtained even if the detachment has been in existence for a considerable period, up to six months. With ruptures of more than one year's duration, the retina may become reattached, but the visual result is poor. (7 tables) Ray K. Daily.

Ursin, K. V. **Retrolental fibroplasia.** Acta ophth. **30**:453-460, 1952.

Twelve brief case histories of retrolental fibroplasia from the eye clinic at Helsinki are reported. In two the diagnosis was confirmed histologically.

Ray K. Daily.

Wirth, Alberto. **The synchronization of impulses in the clinical retinogram.** Acta ophth. **30**:399-402, 1952.

This study deals with qualitative variations and atypical waves encountered clinically in pathologic conditions. A variation in the b-potential (illustrated) was encountered by the author in cases of retinitis pigmentosa and retinal detachment. This small wave probably represents the activity of surviving retinal elements; the registrations of their activity is made possible by their occurrence in areas favorable to registration by the apparatus. In cases with lack of response to the normal stimulus such atypical waves indicate activity in some surviving retinal elements. The absence of normal responses to illumination in such cases may be explained by assuming that the reaction is below the capacity of the registering apparatus, or perhaps that the processes which produce the P I and the P II waves neutralize each other. A variation in the b-potential is described in cases of myopic degeneration. This variation is difficult to explain and the author advances the hypothesis that it may be caused by pathologic alteration in the electrical charge of retinal elements. It is suggested that such data may be utilized clinically. In cases of absence of the normal response such atypical waves indicate the presence of surviving retinal elements. In cases with a normal response the atypical waves indicate the presence of abnormal processes. Ray K. Daily.

Zetterstorm, Birgitta. **The electroretinogram in premature children.** Acta ophth. **30**:405-408, 1952.

This study deals with the appearance of the electroretinogram in premature children. The material comprises thirty premature infants which are divided into three groups according to weight at birth. The tabulated data show that in the lowest weight group the electroretinogram appears after a considerably longer time then in children born at term. The degree of maturity of a child seems to be of importance for the appearance of the electroretinogram. After it has appeared it corresponds in type and appearance with that recorded in children born at term. The author advances the hypothesis that the delay in the lowest weight group may be ascribed with probability to the underdevelopment in premature children of the enzyme systems concerned in the response to a light stimulus. (2 figures, 2 tables) Ray K. Daily.

12

OPTIC NERVE AND CHIASM

Feist, S. **Tabetic atrophy of the optic nerve treated with implantation of pla-**

centa. Klin. Monatsbl. f. Augenh. **123**:34-45, 1953.

It is believed that subconjunctival implantation of placental tissue has a vasodilator effect. Seven patients with tabetic optic atrophy were treated and the author thought he saw some improvement in a few of them. (4 figures, 35 references)

Frederick C. Blodi.

Kenney, J. A., Jr., and Curtis, A. C. **Treatment of syphilitic optic atrophy by penicillin, with and without therapeutic malaria.** Am. J. Syphilis **37**:449-457, Sept., 1953.

The authors treated 37 patients who have syphilitic optic atrophy with penicillin alone and combined with tertian malaria. Their results are in accord with those of Moore who concluded that patients with visual acuity better and 6/20 tend to achieve arrest of the optic atrophy and in those with worse vision the atrophy tends to progress. In this small series all patients can be considered as a fundamentally penicillin-treated group. The addition of therapeutic malaria did not seem to add enough to the effect of the penicillin to warrant its use. Irwin Gaynon.

Palich-Szanto, O. **Pseudoglaucoma.** Klin. Monatsbl. f. Augenh. **123**:20-30, 1953.

The usual course is calcification of some intracranial arteries which precipitate a descending atrophy of the optic nerve. The atrophy is caused by a decreased blood supply to the nerve. Out of four cases only one showed intracranial atherosclerosis. The field defects are often hemianopic and not the characteristic ones found in glaucoma. The intraocular pressure is always normal. (16 figures, 25 references) Frederick C. Blodi.

Vail, Derrick. **Pseudo-glaucoma—a nutritional disease of the optic nerve.** Quart. Bull. Northwestern Univ. M. Sch. **27**:74-77, 1953.

The author discusses the various explanations for the findings in pseudoglaucoma, notably: primary optic atrophy superimposed on a deep physiologic cup (de Wecker and Elschnig); primary cavernous atrophy of the optic nerve (Schnabel); the giving way of a poorly developed (glial supported) lamina cribrosa (Fuchs); a sequel of toxic neuritis (Gradle); and pressure on the optic nerve by calcified carotid arteries. He believes that occlusion of the small nutrient vessels of the nerve lead to cavernous atrophy of the disc and the production of the cup and excavation even when the intraocular pressure is normal or below. Furthermore he cites cases where progress of glaucomatous changes continues after surgery has reduced ocular hypertension to normal, and cases of pseudoglaucoma accompanied by vascular degeneration. Other causes may be tumors near the chiasm, congenital anomalies of the disc, and syphilitic optic nerve atrophy. He then presents two cases of pseudoglaucoma, accompanied by vascular sclerotic changes. Harry Horwich.

13

NEURO-OPHTHALMOLOGY

Bardram, M., and Moller, H. U. **Diagnosis of tumors in the anterior and middle cranial fossa.** Acta ophth. **30**:65-94, 1952.

Twelve cases of suprasellar tumor are reported. It is emphasized that the ophthalmologist is frequently the first to see the patient, and that because of the central scotoma the diagnosis is frequently confounded with that of retrobulbar neuritis or multiple sclerosis. With the aim of an early diagnosis and early treatment, it is urged that arteriography should be

performed early in every case of suspected tumor. (14 figures)

Ray K. Daily.

Kearns, T. P., and Wagener, H. P. **Opthalmologic diagnosis of meningiomas of the sphenoidal ridge.** Am. J. M. Sc. **226**:221-228, Aug., 1953.

The authors reviewed 106 surgically and pathologically verified meningiomas of the sphenoidal ridge from the files of the Mayo Clinic. The average age of the patient was 47, the youngest being a woman 25, and the oldest a man 64. Sex incidence showed 72 percent women. Of the 106, 73 had ocular complaints, 26 had ocular signs, and only 7 had no ocular findings. Visual field defects were found in 60. Proptosis occurred in 34, this being the most common ocular complaint next to visual loss. Papilledema was seen in 46 cases, and 42 showed pallor of one or both discs, five showing the Foster-Kennedy syndrome. Pupillary change as the only ocular finding was seen in but one case. Weakness of ocular muscles occurred in 15; only 7 of these were due to cranial nerve involvement, the others were due to the proptosis. The abducens was the nerve most often involved. Roentgenographic abnormalities were seen in 82 cases, 53 with sufficient changes for diagnosis by this means alone, the most characteristic finding being osteomatous thickening in the region of the wings of the sphenoid. They then describe a typical "composite" case. Harry Horwich.

Koistinen, Aunc. **Recurrent ophthalmoplegia and unilateral papilledema in a case of hyperostosis frontalis interna.** Acta ophth. **30**:421-428, 1952.

A brief review of the literature on hyperostosis frontalis interna is followed by the report of a case in a woman, 38 years old. From the age of 24 years she had had recurrent attacks of ophthalmoplegia, first at 3 to 4 year intervals, and later more frequently. Left-sided papilledema was found in the course of the ocular examination, and hyperostosis frontalis interna was revealed by the X-ray examination of the skull. The ocular symptoms are attributed to new bone formation in the sphenoidal fissure. The recurrent attacks of ophthalmoplegia are explained by changes in the vascular volume, and the persistent papilledema by a continuous venous stasis in the stenosed sphenoidal fissure. There were no other neurological symptoms. (2 roentgenographs)

Ray K. Daily.

14

EYEBALL, ORBIT, SINUSES

Engel, F. L. **An unusual case of malignant exophthalmos and postoperative hypothyroidism complicating Graves' disease.** J. Cl. Endocrin. **13**:1132-1139, Sept., 1953.

Nine months after a patient was given thyroid medication for general fatigue she developed hyperthyroidism. In spite of medication with propylthiouracil and iodine the exophthalmos increased with loss of color vision. A thyroidectomy was performed and the eye symptoms became worse. The patient was given X-ray treatment in the pituitary but the vision in the right eye deteriorated. She developed a full blown myxedema and when she resumed thyroid medication she improved remarkably. After 34 months her vision has improved and the exophthalmos has diminished slightly. (3 figures)

H. C. Weinberg.

Malatesta, C. **Unusual subacute traumatic frontal mucocele invading the orbit.** Boll. d'ocul. **32**:233-236, April, 1953.

A farmer, 70 years old, was injured by

the horn of a cow, and was seen eight months later with an extensive defect of the orbital roof permitting the mucous membrane of the left frontal sinus to protrude widely into the left upper lid. During the operation contact with the dura mater, and pachymeningitis, was found. A good cosmetic functional result was achieved. (2 photographs, references)

K. W. Ascher.

15

EYELIDS, LACRIMAL APPARATUS

Bergler, K. **Reconstruction of the canaliculus according to Thiel.** Klin. Monatsbl. f. Augenh. **123**:53-58, 1953.

The original operation of Thiel (Klin. Monatsbl. f. Augenh. 108:576, 1942) was modified in two cases. This operation, a plastic repair of the canaliculus was designed as a tube of mucous membrane around a plastic probe. In two cases the author has done this operation without a probe. This simplified the operation considerably. Changes in the form and location of the caruncle are important factors in the movement of the tears.

Frederick C. Blodi.

Gronvall, Herman. **Sebacious cysts in the region of the eyes.** Acta ophth. **30**:19-31, 1952.

An analysis of 375 case histories of sebacious cyst of the face shows that sebacious cysts are seen with equal frequency in both sexes; that the majority of the patients were between the ages of 60 and 80 years; that the relative frequency rises gradually up to the age of 50 and then rapidly; that the tendency to bilateral occurrence increases with advancing age, and then the cysts are most frequently multiple; that the number of cysts on the left side is greater than on the right; that the cysts show a predilection for the skin around the eyes and the proximal part of the root of the nose, particularly in the inner angle of the eyelid, and then especially just above the internal palpebral ligament; that the distribution of the cysts around the two eyes is usually symmetrical; that the areas affected are the same as those involved by comedones; and that they follow approximately the cleft lines of the skin, and are of about the same size in the various regions on the face. (8 figures, 6 tables)

Ray K. Daily.

Junghannss, K. **Rethotelsarcoma on the eye.** Klin. Monatsbl. f. Augenh. **123**:95-98, 1953.

Rethotelsarcoma in the right eye in a 65-year-old man with generalized sarcomatosis is described. Another tumor was in the lacrimal sac of the same eye. The primary tumor was apparently in the vocal cord. (1 figure, 17 references)

Frederick C. Blodi.

Kaldeck, Robert. **Ocular psoriasis.** Arch. Derm. & Syph. **68**:44-49, July, 1953.

Eleven cases are reported in which there is psoriasis of the lids and in some of which there is involvement of the bulbar conjunctiva and cornea. In only two cases did the patient show ocular psoriasis alone and of these one had a long standing dermal psoriasis. The other patient had a history of psoriasis in the family. Treatment with mild yellow mercuric oxide and X ray or radium is recommended. Ocular psoriasis is twice as frequent in the male as in the female. (2 figures)

H. C. Weinberg.

King, J. H., Jr., and Forrest, R. L. **Vaccinia of the eyelids; treatment by aureomycin in a second case.** J.A.M.A. **153**:31-32, Sept. 5, 1953.

A six-year-old girl developed vaccinia of the right eyelid six days after she was vaccinated on her left upper arm. Penicillin intramuscularly and streptomycin ointment locally did not alter the progress of the lesion. Oral aureomycin 250 mg. and aureomycin solution locally every four

hours and irrigation with potassium permanganate (1/100,000) and oral ascorbic acid 50 mg. twice daily were given. The child's eye improved within 24 hours and after 96 hours the ulcers and inflammation had healed completely with no evidence of scarring two weeks later. (2 figures)

H. C. Weinberg.

Papst, W. **Transplantation of supramid tubes in stenosis of the lacrimal duct.** Klin. Monatsbl. f. Augenh. **123**:58-69, 1953.

This is a survey of 26 cases operated on in Kiel. A good result was achieved in 8 patients, 4 had an acute dacryocystitis soon after the operation, 9 again had lacrimal stenosis a few weeks after the operation and in 6 the tube was extruded into the nose. These results are therefore far below the usual excellent end-results of dacryocystorhinostomy. (12 references)

Frederick C. Blodi.

17

INJURIES

Stenius, S. **Some remarks regarding the localization of intraocular foreign bodies.** Acta ophth. **30**:115, 1952.

The author comments on the failure of the Stenius-Ribbing locator to find general acceptance.

Ray K. Daily.

Werner, Sigurd. **Injuries to the eyes of children.** Acta ophth. **30**:97-104, 1952.

Two hundred and fifteen case histories of hospitalized patients with ocular injuries are reviewed. Ten were bilateral. The injuries were 50 percent more common during the latter half of the year. The commonest causes were perforations with sharp instruments, and explosions. In more than one third of the cases the damage was self-inflicted, and in an almost equal number the damage was inflicted by a playmate. Of the 225 eyes 53 were enucleated or exenterated, and in 28 vision was lost. The author calls attention to the need for education for prevention of ocular accidents. (5 tables)

Ray K. Daily.

18

SYSTEMATIC DISEASE AND PARASITES

Weizenblatt, S. **Ophthalmomyiasis externa; larval conjunctivitis in socket.** A.M.A. Arch. Ophth. **50**:79-80, July, 1953.

Larvae of the black blow fly were identified as contaminants in a socket after an enucleation for retinoblastoma. After removal of the maggots, the secondary infection was successfully treated with streptomycin and penicillin.

George S. Tyner.

19

CONGENITAL DEFORMITIES, HEREDITY

Carroll, F. D., and Haig, C. **Congenital stationary night blindness without ophthalmoscopic or other abnormalities.** A.M.A. Arch. Ophth. **50**:35-44, July, 1953.

The pedigree of a family with this disease is reported. Functional tests indicated a complete lack of rod function. An unaffected child of parents with this type of night blindness never carries the disease and none of the descendents of such a child are affected. The disease is a dominant characteristic.

George S. Tyner.

Oravisto, Terttu. **Hereditary hemorrhagic telangiectasis as an eye disease.** Acta ophth. **30**:447-451, 1952.

The case reported is that of a woman, 35 years old, whose father was subject to attacks of nasal hemorrhage. She developed a typical clinical picture of hemorrhagic telangiectasis in the form of recurrent nasal hemorrhage and telangiectatic areas on the lips, tongue, face, and hands. She came to the eye clinic because of loss of vision in the left eye, which was found to be caused by choroidal melanoma. The literature on telangiectasis is briefly re-

viewed and it is pointed out that the combination of telangiectasis and melanoma has been reported only once before.

Ray K. Daily.

20

HYGIENE, SOCIOLOGY, EDUCATION, AND HISTORY

Alcala Lopez, Antonio. **A classification table for the etiology of blindness and the statistics on blindness in Malaga.** Arch. Soc. oftal. hispano-am. 13:642-657, June, 1953.

The author proposes a classification of blindness on a scale in which the topographic classification is placed on the ordinate, and the etiologic on the abscissa. There are ten divisions on each line. A statistical study of blindness in Malaga shows a diminution of blindness from infection and ophthalmia neonatorum, and an increase of blindness caused by malignant myopia and trachoma; these two and glaucoma are at present the principal causes of blindness. (10 tables)

Ray K. Daily.

Ophthalmic Miniature

Answer to correspondence of
Dec. 27, 1884

January 15, 1885

Dear Sir:

I have not been able to get the proofs ready for the new edition of "Physiological Optics" but I have collected and arranged the literature which has accumulated in the meantime with the help of my assistant, Dr. Koenig. In this way the beginning of the work is prepared and I shall be able to take care of the proofs personally in the very near future and provide, if necessary, additions and changes. I hope that the work will progress quickly with the exception of a few paragraphs. I figure that we can start printing early in April if you do not want more than one galley per week.

At this time the question arises whether to continue enlarging the old bibliographic references in view of the fact that we already have them on hand. In that case it might be better to put them together at the end in order not to interrupt the text by long lists of references. However, there is so much literature that it might take up five galleys. This may not be as necessary as formerly in view of the now widespread circulation of the yearly reports of the "Medical Sciences." Dr. Koenig who collects and arranges the literature for me has to do practically all the work in any case. I don't know what you think about it from the bookseller's point of view. I would like to review a set of galley proofs in order to be able to work out the fee.

With best regards,

Yours
H. von Helmholtz

P.S. Since the first publication of the "Physiological Optics" a great number of better woodcuts have been published in other books, particularly anatomical pictures, which I would like to incorporate. Usually there are printer's cuts of these first pictures available. Would you mind if I sent you a list of such pictures?

To: Leopold Vohs
Book Editor
Hamburg

From the autograph collection of
Dr. James W. Smith, New York.

NEWS ITEMS

Edited by Donald J. Lyle, M.D.
601 Union Trust Building, Cincinnati 2

News items should reach the editor by the 12th of the month but, to receive adequate publicity, notices of postgraduate courses, meetings, and so forth should be received at least three months before the date of occurrence.

Deaths

Dr. Arcangelo Liva, Los Angeles, California, died August 12, 1953, aged 66 years.

Announcements

California postgraduate courses

The University of California School of Medicine, San Francisco, offers Part II of the didactic resident course in ophthalmology from January through May, 1954. The course, to be presented jointly by the University of California School of Medicine and Stanford University School of Medicine, is organized and presented specifically for residents in training in ophthalmology but there are a few places for practicing physicians interested in a review of the many subjects under discussion.

Requirements for admission are graduation from an approved medical school and license to practice in a respective state. For further information write to:

Dr. Stacy R. Mettier
Head of Postgraduate Instruction
Medical Extension
University of California Medical Center
San Francisco 22, California

Pan-American course

On July 4, 1954, a school of ophthalmology will open in Havana, Cuba, to give instructions in diseases of the eye to qualified physicians. The course will be of two years' duration arranged in four semesters and will consist of theoretical as well as of practical instruction in ophthalmic practice and surgery of the eye. The course is limited to 20 physicians from the American continent, under 30 years of age, who were graduated less than five years ago and have a knowledge of Castilian Spanish and other languages spoken on the American continent. The school will use the facilities of the hospital of the League against Blindness; students will see patients in the dispensary and will take part in operations and other activities. A diploma of graduation will be issued to those who pass the examination satisfactorily.

The cost per semester will be $250; that is $500 per year. Students coming from places other than Havana will have to add room and board. Details of the course, time of registration, and so on may be obtained by writing to:

Dr. Manuel Fariñas Pelletier
Secretario General de la Escuela
Liga contra la Ceguera
Apartado 4053
Habana, Cuba

The creation of the school of ophthalmology was the result of the Pan-American Congress of Ophthalmology and the efforts of Dr. Derrick Vail, Chicago; Dr. Varriere, Montevideo; and Dr. Urrets Zavalia of Cordova, Argentina. One of the purposes is to cement Pan-American friendship. Scholarships for young men are needed and it is hoped that $3,000 can be raised.

Call for papers for San Francisco meeting (June 21-25, 1954)

The 1954 meeting of the Section on Ophthalmology will be held in San Francisco, June 21-25, 1954. Again the section will meet simultaneously with the Association for Research in Ophthalmology.

Anyone wishing to present a paper before the Section on Ophthalmology is urged to communicate with Dr. Harold G. Scheie, secretary of the section, 313 South 17th Street, Philadelphia 3, Pennsylvania, as soon as possible. A title and abstract must be in his hands by January 1, 1954.

Anyone wishing to apply for space in the scientific exhibit of the American Medical Association should contact Dr. William F. Hughes, Jr., 1853 West Polk Street, Chicago 12, Illinois.

Anyone wishing to present a paper before the Association for Research in Ophthalmology should communicate with Dr. Lorand V. Johnson, secretary of the association, 10515 Carnegie Avenue, Cleveland 6, Ohio, as soon as possible.

Members are reminded that their contributions to the Knapp Fund are now due and payable to Dr. Parker Heath, 243 Charles Street, Boston, Massachusetts. A $10.00 contribution entitles you to a volume of the 1953 *Transactions*. Contributions in excess of $10.00 are invited.

Eye-Bank in Atlanta

The Ponce de Leon Eye, Ear, Nose, and Throat Infirmary, Atlanta, Georgia, affiliated with the national Eye-Bank for Sight Restoration, Inc., is offering its services for those who wish to donate their eyes to the Eye-Bank at time of death. Physicians may obtain the necessary forms for this service from the infirmary. The infirmary will make all arrangements after notification by the physician.

New Orleans convention

Featured at the Mid-Winter Convention of the New Orleans Academy of Ophthalmology, meeting February 1 through 5, 1954, will be a symposium on "Infectious diseases of the eye." Those leading the symposium will be: Dr. Phillips Thygeson, San Jose, California, "Virus infections of the lids and

conjunctiva"; "Bacterial infections of the conjunctiva"; Dr. Trygve Gundersen, Boston, "Infectious diseases of the cornea"; Dr. Alson E. Braley, Iowa City, Iowa, "Allergy and drug sensitivity"; Dr. Hugh Ormsby, Toronto, "Infectious diseases of the lids, lid margins, and lacrimal apparatus"; Dr. Michael J. Hogan, San Francisco, "Infectious diseases of the episclera, sclera, and uveal tract"; "Surgical infections"; Dr. Gustav Bahn, New Orleans, "Infections of the orbit"; Dr. James H. Allen, New Orleans, "Ocular bacteriology."

The program will also include round-table discussion periods. The registration fee of $75.00 includes associate membership in the academy for the year, 1954.

Communication aids for the blind

Better ways for the blind to read, walk, and communicate were discussed by research authorities in the field at a symposium on "Communication aids for the blind" held on December 28th, in Room E, Mechanics Building, Huntington Avenue, Boston, Massachusetts, in conjunction with the 120th meeting of the American Association for the Advancement of Science.

Societies

Nassau meeting

Dr. Alston Callahan, Birmingham, showed a movie on "Lacrimal canaliculus" at the November 23rd meeting of the Nassau Ophthalmological Society. Dr. Frank Bussey spoke on "An industrial eye program." Dr. Melvin Harbater reported a case of "Scleromalacia perforans"; Dr. Jesse Michaelson, "Bilateral thrombosis of central retinal arteries"; Dr. Gerard M. Branower, "Olfactory groove tumor." Dr. Charles Taintor and Dr. Peter H. Ballen presented new instruments.

Reading report on Academy meeting

At the 137th meeting of the Reading (Pennsylvania) Eye, Ear, Nose, and Throat Society, the following members of the society, who were present at the meeting of the American Academy of Ophthalmology and Otolaryngology, Chicago, reported briefly on courses and lectures attended: Dr. Gaylord W. Ojers, Dr. Benjamin F. Souders, Dr. John J. Penta, Dr. Harold L. Strause, Dr. Arthur A. Bobb, Dr. Paul Craig, Dr. Robert Muschlitz, Dr. Robert E. Shoemaker, Dr. James H. Parker, Jr.

Dallas Academy program

The 1953-1954 program of the Dallas Academy of Ophthalmology and Otolaryngology opened with a symposium on "Dermatology in ophthalmology and otolaryngology," at which Dr. Shelton Blair, Dr. Ludwig Furchgott, and Dr. Ronald Burnside spoke. The honor guest at the second meeting was Dr. Kinsey M. Simonton, Rochester, Minnesota. The program for the third session was a panel discussion led by Dr. Otto Grunow, Dr. Ray Harrington, and Dr. Edward Newell, on "Radiology in ophthalmology and otolaryngology."

Future sessions will be held on:

Tuesday, January 5, 1954

5:30 p.m.—Clinical Conferences, Parkland Hospital
6:30 p.m.—Dinner honoring speakers, Melrose Hotel
7:30 p.m.—Symposium: Blood dyscrasias in ophthalmology and otolaryngology
Dr. Joe Hill—Hematology
Dr. Dudley Singleton—Otolaryngology
Dr. Lester Quinn—Ophthalmology

Tuesday, February 2, 1954

Honor Guest: Dr. Brittain F. Payne, New York, New York
1:00 p.m.—Luncheon honoring Dr. Payne, Melrose Hotel
2:00 p.m.—Lecture: Extraction of complicated cataracts
6:30 p.m.—Dinner honoring Dr. Payne, Melrose Hotel
7:30 p.m.—Lecture: Complications following intraocular operations

Wednesday, February 3, 1954

1:00 p.m.—Luncheon honoring Dr. Payne, Melrose Hotel
2:00 p.m.—Lecture: Operations for retinal detachment
6:30 p.m.—Dinner honoring Dr. Payne, Melrose Hotel
7:30 p.m.—Lecture: Plastic operations on the eyelids

March 15-18, 1954

The Dallas Southern Clinical Society Meeting
Guests:
Otolaryngology: Dr. Anderson C. Wilding, Dr. Walter P. Work
Ophthalmology: Dr. Alson E. Braley, Dr. Dwight Townes

Tuesday, April 6, 1954

5:30 p.m.—Clinical Conferences, Parkland Hospital
6:30 p.m.—Dinner honoring speakers, Melrose Hotel
7:30 p.m.—Program: Medical illustration and photography
Prof. Lewis Waters, Southwestern Medical School; Dr. Fred Newton—Movie on cataract surgery; Dr. Donald Corgill—Movie on mastoidectomy.

Tuesday, June 1, 1954

5:30 p.m.—Clinical Conferences, Parkland Hospital
6:30 p.m.—Dinner honoring speakers, Melrose Hotel
7:30 p.m.—Program:
Dr. W. P. Anthony, Fort Worth: "The right to look human."
Dr. James H. Scruggs, Waco: "Metabolic exophthalmos."

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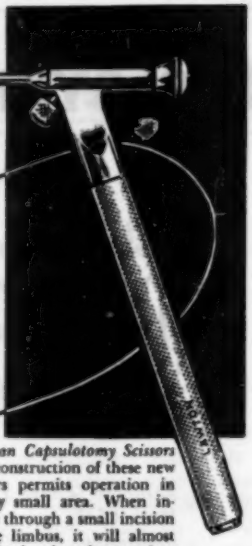

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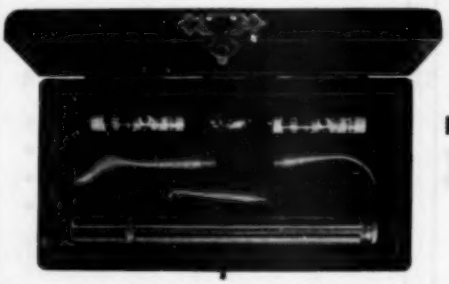

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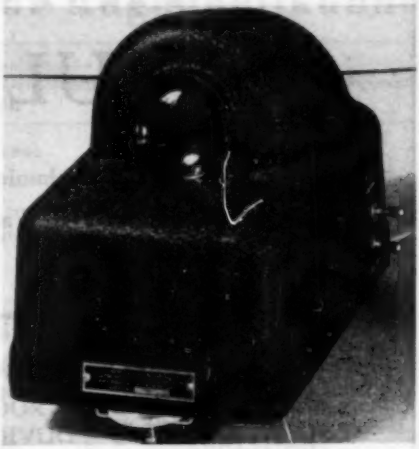

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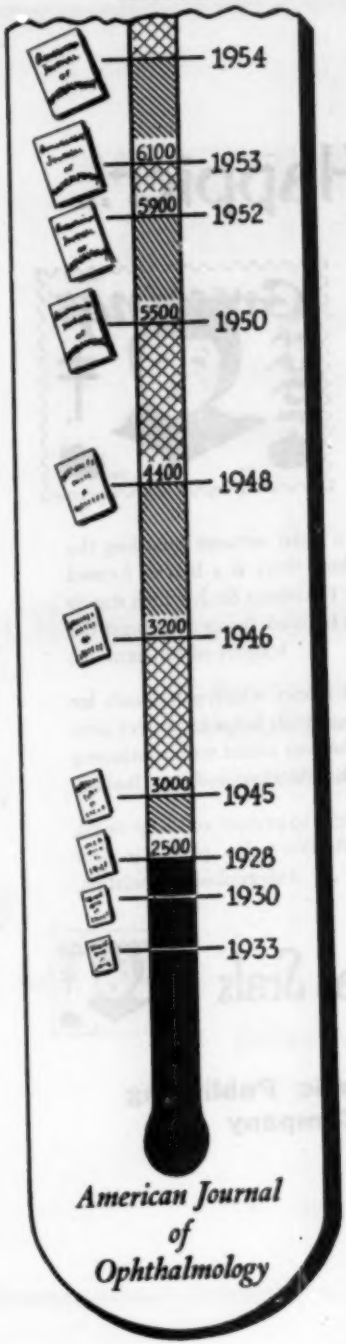
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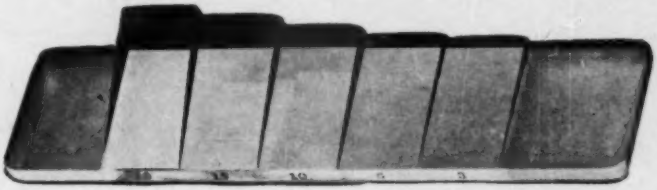

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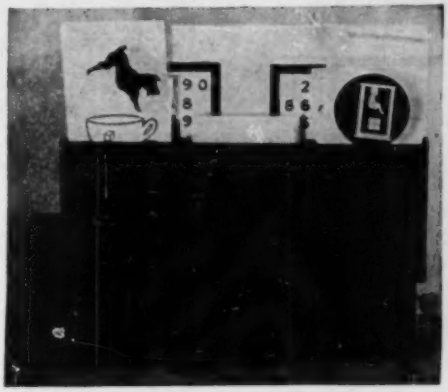

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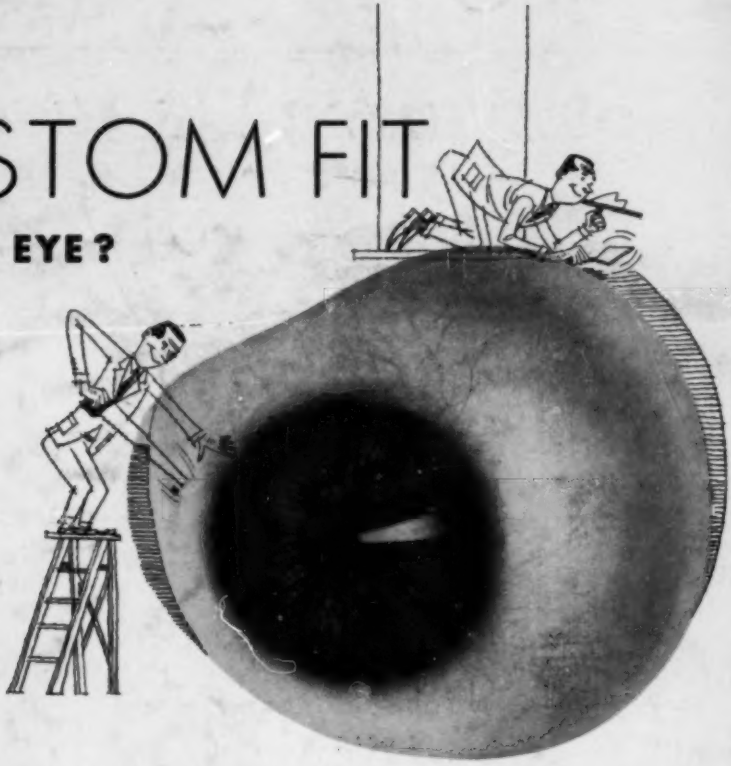

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